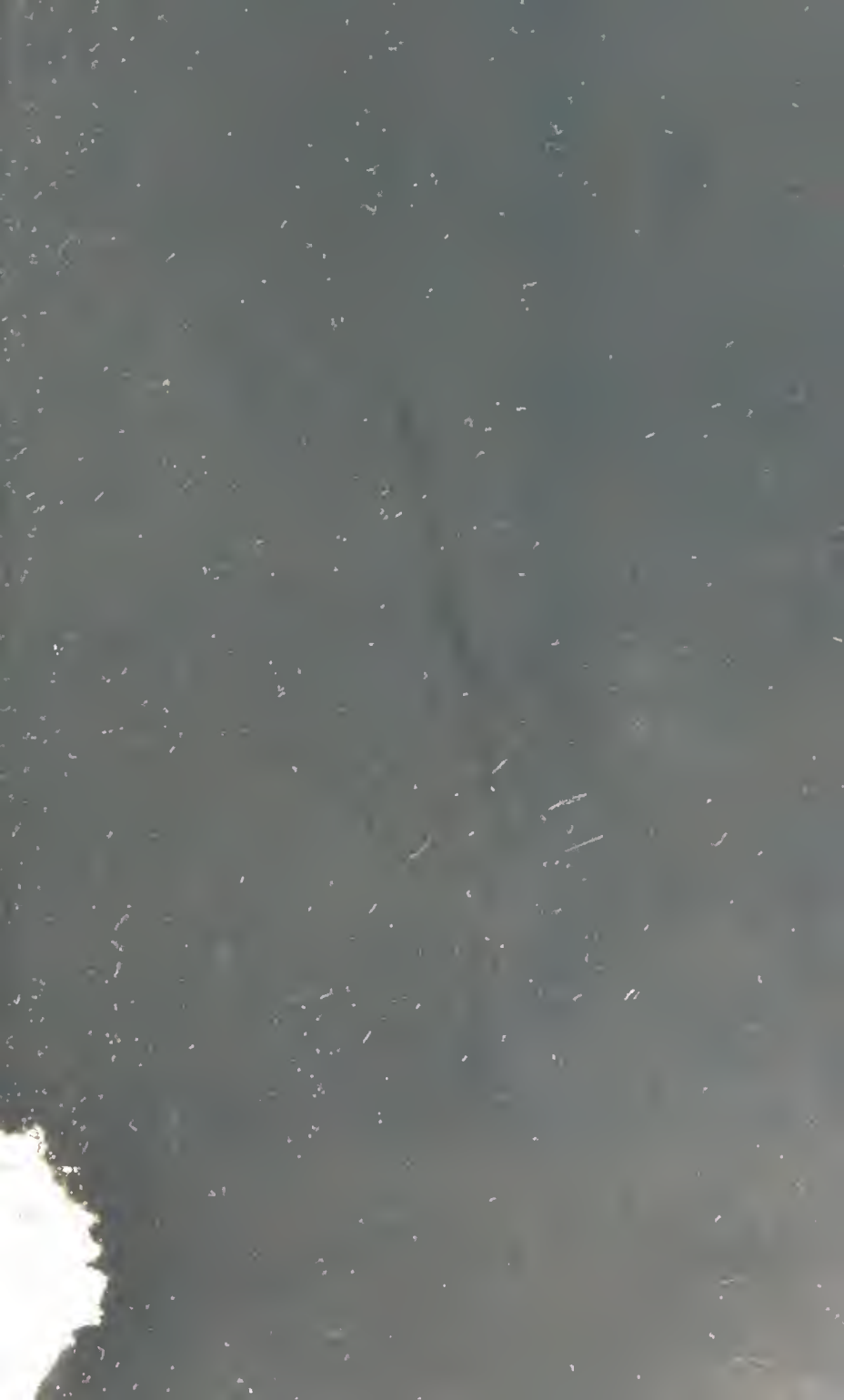
Pulmonary Tuberculosis

CARLO RUATA, M.D.



PULMONARY TUBERCULOSIS.

PULMONARY TUBERCULOSIS:

ITS PREVENTION AND CURE.

WITH APPENDIX CONCERNING
THE BRITISH CONGRESS ON TUBERCULOSIS, 1901.

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PREFACE.

This small work has been written for the purpose of making known certain facts calculated to throw a greater light on the origin of pulmonary tuberculosis; to show how it may be possible to prevent its manifestation, and how it may be cured with certainty, if the treatment is undertaken at a time when the nature and extent of the lesions in the lungs have not rendered the case hopeless.

Since every rational method either of prevention or cure must be founded on the anatomical and physiological conditions, and the ensuing pathological changes, I have thought it well to give first an abridged description of such conditions, and especially of those parts which are most important in influencing the progress of the disease.

I am convinced that in the great conflict that has now begun with tuberculosis, medical science must appeal to all who

are interested in it, so that they may make themselves fully aware of what the disease is, what means there are of combating it, and may in good time have recourse to them either to cure or prevent the illness. This conviction has prompted the endeavour to write this volume in as far as possible non-technical language that may be understood by all.

I hope that whoever reads these pages will not regret the hours spent in perusing them, and it will be sufficient reward if I succeed in inspiring my readers with those principles of self-protection and of action that we all ought to cultivate in order to combat successfully the greatest scourge of our times.

CARLO RUATA

PERUGIA, ITALY,
September, 1901.

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PULMONARY TUBERCULOSIS:

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CHAPTER I.

INTRODUCTORY.

OF all the forms of tuberculosis, that of the lungs is by far the most deadly as well as being probably a fruitful source of the other forms. It has been calculated that a person infected with pulmonary tuberculosis emits on an average, by expectoration alone, about 350,000,000 in the course of twenty-four hours of Koch's bacilli. The deaths due to this cause in Italy are at least 80,000 a year. Calculating this illness to have an average duration of three years, we must conclude that the number of consumptive persons among us is certainly not less than 250,000. Putting this number in relation to the number of bacilli emitted by every sick person, we understand how it is that tuberculosis is so widely spread. The other forms, such as lupus, scrofula, tabes mesenterica, fungous arthritis, and

tuberculosis of the bones, are infinitely less perilous to the public health; therefore, if the pulmonary form could be arrested, the rest would prevail to a far less extent, and possibly would disappear altogether.

However, we are not called upon to believe that the peril of contracting tuberculosis is so great as the figures we have stated seem to indicate. Were it so, we should all die of consumption. I will show later on that, notwithstanding the many bacilli that surround us, the danger is relatively small and becomes trifling if we take some precautions suggested by the nature of the facts and by the anatomical construction of our breathing apparatus.

Before we proceed, let us notice some anatomical facts which bear on what we shall say hereafter concerning the disease.

ANATOMICAL AND PHYSIOLOGICAL CONDITIONS.

The nasal fossæ are lined with a layer of cylindrical and ciliated cells which are constantly kept moist by the mucus and serum furnished by the numerous mucous and serous glands which exist in this region.

The trachea is also lined with a treble layer of epithelial cells, the most superficial of which are likewise ciliated and kept moist in the manner

mentioned above. The trachea is from 11 to 12 centimetres long and has a diameter of 2 or $2\frac{1}{2}$ centimetres, and at the level of the third dorsal vertebra it subdivides into two large bronchi right and left, each of which serves to carry the air to the corresponding lung.

These two bronchi subdivide themselves in their turn repeatedly in the following way: (1,) The subdivision proceeds symmetrically and dichotomously until the smallest have reached the diameter of about 4 millimetres; (2,) When this size

FIG. 1.

Terminal branch of a bronchial tube with its infundibula, alveolar canals and alveoli obtained from the lung of a monkey injected with metallic mercury (× 15 diameters.): (*a*, Terminal bronchial branch; *b*, Infundibula and alveoli; *c*, Alveolar canals and alveoli. If to these we add: (1, A little branch of the pulmonary artery that accompanies the terminal branch of the bronchus and enters it, sub-dividing itself into tiny capillary tubes that line every alveolus; (2,) A corresponding branch of pulmonary vein found by the re-union of the aforesaid capillary vessels and coming out carrying the blood from the alveoli; (3,) An accompanying nerve with similar ramifications; and (4.) Some connective tissue binding together the canals, infundibula, vessels and nerves, we form the whole pulmonary lobule.

is reached they proceed in straight lines, gradually diminishing in size and sending off laterally and alternately still further ramifications; (3,) These again subdivide themselves dichotomously until

they have reached the size of about a fourth of a millimetre; finally terminating in canals or alveolar passages which together constitute the pulmonary lobules.

The *pulmonary lobule* may be considered a miniature lung. Every alveolar canal is slightly larger than the bronchus from which it starts, and

FIG. 2.

Alveoli of an infundibulum from the inside: *a*,) Alveolus lined with pavement-epithelium cells not provided with cilia, whilst the epithelial cells of the bronchi are cylindrical and furnished with vibratile cilia; *b*, Alveolus deprived of epethelium showing its surface formed of connective elastic tissue; *c*,) Alveolus with its capillary net

subdivides at once dichotomously two or three times, to terminate in as many somewhat bulbous extremities, which are called infundibula. On the inner surface of the alveolar canals and infundibula are small saccular dilatations called *air-sacs*, *air-cells*, *air-vesicles*, or *alveoli*, in size

about three-twentieths of a millimetre. The alveolar canals and infundibula are principally formed of fibres of elastic tissue with a few unstriated muscular fibres. The alveoli are also formed of a very thin membrane of elastic tissue which supports a layer of capillary vessels. Thus we see that every pulmonary lobule is formed by the sub-division of the alveolar canals with a great number of alveoli opening on these canals.

The pulmonary lobules vary in size from 2 to 4 millimetres. The great pulmonary mass is made up of an endless number of these lobules closely adhering to each other, though in the fœtus they may be easily divided.

Every terminal bronchus is accompanied by a small ramification of the pulmonary artery which subdivides into very minute capillary vessels on the surface of the alveoli, of the infundibula, and of the alveolar canals. These vessels form a very close network and are extremely thin, their calibre being 5 or 6 micro-millimetres (5 or 6 thousandths of a millimetre). Here the air undergoes its changes, the blood yielding up carbonic acid and taking the oxygen necessary for the oxygenation of the tissues.

Following now the whole bronchial tree from the trachea to the infundibula and alveoli, let us note some facts of great importance. The epithelial ciliated cells that we have seen in the

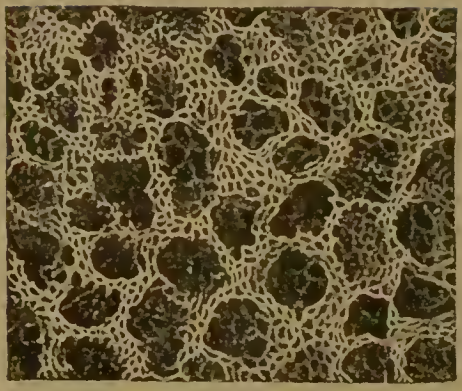


FIG. 3.
The pulmonary alveoli in man (× 70 diameters.

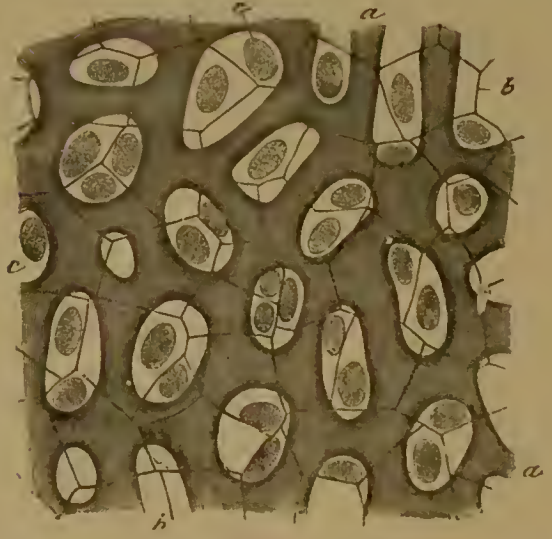


FIG. 4.
Inner side of the alveoli greatly enlarged : (*a*,) Net of capillary vessels : (*b*,) Pavement-epithelium cells : (*c*, Nuclei of the pavement cells.

nasal fossæ and in the wind-pipe extend in the same way throughout the length of the several bronchi to the alveolar canals. In the latter, as well as in the infundibula and alveoli, the epithelial cells are deprived of vibratile cilia.

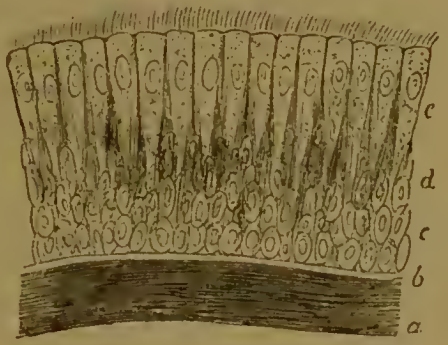


FIG. 5.

Ciliated epithelium of the human trachea (× 350 : (*a*, Layer of longitudinal elastic fibres; *b*, Basement membrane; (*c*,) Round, deep cells; (*d*,) Intermediate elongated cells; *e*, External layer of cylindrical cells completely developed and bearing vibratile cilia.

The office of these cilia is of great importance. They vibrate constantly and determine a flow of the mucus from bottom to top, serving to eliminate from the air passages any foreign substance that may have been drawn by the act of breathing on to some part of the bronchial mucous membrane.

Another important fact is this: The trachea is mainly constructed of fibro-elastic membrane, in the layers of which are to be found rings of cartilaginous tissue, making it a stiff tube always open. These rings, which even in the trachea are incomplete on the posterior side, become much more

so the farther one descends into the lower bronchi, till, reaching the middle, one finds but little bits or irregular patches of cartilage strewn along the surface. In the minor bronchi, that is to say in those that terminate in the alveolar canals, the cartilaginous tissue disappears altogether.

It follows that, while the larger bronchi are always open tubes, the minor ones are probably collapsible, as a writer has observed, and open only by inspiration and expiration.

Where the two bronchi, into which the wind-pipe divides, enter the lungs to form the great bronchial tree, the pulmonary artery enters also, and it subdivides in the same manner until the furthest ramifications, forming the thick capillary net we have mentioned, are reached. These capillary ramifications re-unite in the same manner to form the pulmonary veins which carry the blood enriched with oxygen to the two left cavities of the heart, leaving the lungs at the same spot at which the pulmonary artery entered them. This point is called the root of the lung.

An enormous quantity of lymphatic vessels commence in the minute interstices that are to be found throughout the lung in the connective tissue that unites the alveoli, the infundibula, the bronchial tubes, the blood-vessels and the pleura. These vessels pass in towards the root of the lung to reach the bronchial glands, and

afterwards proceed—the left into the thoracic duct, and the right into the right lymphatic trunk, the direction of these lymphatic vessels being thus from the periphery of the whole lung towards the root.

During inspiration many impure substances are introduced into the lung, which may also be eliminated by the current of the lymph running through the lymphatic vessels. We have a proof of it in the fact that coal-workers often have black bronchial lymphatic glands owing to the coal dust that the lymphatic vessels have carried to them.

Finally, we remember that the two lungs occupy almost the entire thoracic cavity formed by the ribs, and, with their apices, reach three inches higher than the collar bone on both sides, while their bases lie on the diaphragm. Each lung is enveloped by a serous membrane, the pleura, one layer of which adheres closely to its surface, and provides it with its smooth covering, while the other is attached to the inner surface of the chest-wall, leaving a space (sac of the pleura) between the two layers (the parietal and the visceral), allowing the lungs to glide easily in the action of breathing.

CHAPTER II.

AIR CHANGES IN THE LUNGS.

CONSIDERING the manner in which the exchange of air takes place in the lungs, an important fact should be noted. During inspiration, through the upward movement of the ribs and the lowering of the diaphragm, the thoracic cavity is enlarged, and air must necessarily rush into the lungs to equalize the pressure between the outside air and the diminution that occurs in the inside. But we must remember that air reaches the infundibula through the direct impulse of inspiration only with difficulty ; the air that enters the infundibula and alveoli does so by diffusion. Moreover, no effort of expiration, however powerful, can force the alveolar air beyond the lesser bronchi, which indicates that the oxygen of the air reaches them by diffusion according to the following principle : If two chambers, containing a mixture of gas in unequal proportions, are put into communication with one another, a movement of gas called diffusion begins at once and continues until both are thoroughly incorporated. Let us suppose that one

chamber contains a great quantity of oxygen and little carbonic acid, and the other much carbonic acid and little oxygen ; the oxygen will pass from the former to the latter, and the carbonic acid from the latter to the former until the two chambers contain the gases in similar proportions. This movement is due to the elastic property of gas, which compels its constant expansion until the space containing it is entirely filled.

Now the quantity of oxygen, nitrogen and carbonic acid varies as follows in a hundred volumes of air inspired and expired :—

	Oxygen.	Nitrogen.	Carbonic Acid.
Inspired air contains ...	20·96	79	·04
Expired air contains ...	16.03	79	4·4

Therefore the air of the alveoli always contains a smaller proportion of oxygen than the atmospheric air ; hence, at every inspiration the oxygen enters the alveoli by diffusion. Alveolar air also contains a larger quantity of carbonic acid than the atmosphere, and therefore the carbonic acid of the alveoli continually diffuses from within the lesser bronchi to the atmosphere outside.

From the alveoli the oxygen penetrates the blood, and the carbonic acid of the blood enters the alveoli, partly by the same law, but more because chemical changes occur which need not detain us here.

The figures quoted above show that the nitrogen undergoes but very slight changes, and we therefore see that while some gases are not absorbed by the blood in the alveoli, there are others which pass from the blood in minute quantities into the alveoli to be eliminated through the bronchioles.

This leads us to ask: When we inhale a gas, are we sure it reaches the very utmost bronchiole and alveoli? And the answer is said to be uncertain. Of some gases we are perfectly sure; for instance, pouring a little ether or chloroform on a pocket handkerchief and inhaling it, we feel the effects on the brain after the first inhalation, proving that the ether and chloroform pass into the alveoli and thence, through the blood, to the brain. And the same happens with some other gases, although on this point all doubt is not yet cleared up. But it is probable that all gases do reach the farthest recesses of the lungs by diffusion, and only some of them can be absorbed by the blood in the capillaries.

Nor can we fully reply to another question: If a gas circulates in the blood, when it reaches the capillary vessels of the pulmonary alveoli, in what proportion is it discharged by means of the alveoli to be eliminated and brought into the atmosphere? For instance, when creasote is administered to a patient by clyster, after a short time the patient has a taste of creasote in his mouth, showing probably that the creasote has been partly yielded

up by the blood of the lungs' capillaries to the air in the alveoli, and from these it has reached the mouth. On the other hand, the elimination of creasote might have taken place through the salivary glands, producing the same effect in the mouth.

It is, however, certain that the elimination of creasote through the alveoli takes place, if at all, in only the slightest quantity, for it continues to circulate in the blood, to be eliminated by the natural emunctories, and especially by the kidneys, giving the urine a brown or smoky appearance, if in large quantities.

Finally, in expired air, not only is there a great quantity of carbonic acid, but there are also many impurities, for the most part unidentified, which are infinitely more injurious than carbonic acid.

These impurities, not reckoning organic matters that remain in the mouth among the teeth and putrefy, corrupt the air we breathe very considerably. In large cities, in highly-crowded places, the air contains in abundance many varieties of micro-organisms which rapidly putrefy the organic constituents of the expired air. Some of the elements derived from putrefaction seem to be endowed with highly poisonous properties; other elements equally poisonous, of the nature of the ptomaine, are eliminated with the expired air as direct results of the act of breathing. These impure matters,

though in very small proportions, are deadly in their effects, and are those that give that peculiar foul smell to the air of rooms which have been kept shut, which is felt if we remain for a while in a room without opening the windows. We know that the number of expirations of a sound person in one day is about 28,000, and in a person affected with pulmonary tuberculosis who emits an immensely larger quantity of micro-organisms, this number may increase to 40,000 and even 50,000. We see at once, therefore, that the quantity of such hurtful matter becomes considerable in the space of twenty-four hours; hence the great importance of change of air for this class of patients.

CHAPTER III.

PULMONARY CHANGES IN TUBERCULOSIS.

By whatever route the bacillus of tuberculosis (the only cause of pulmonary tuberculosis) reaches

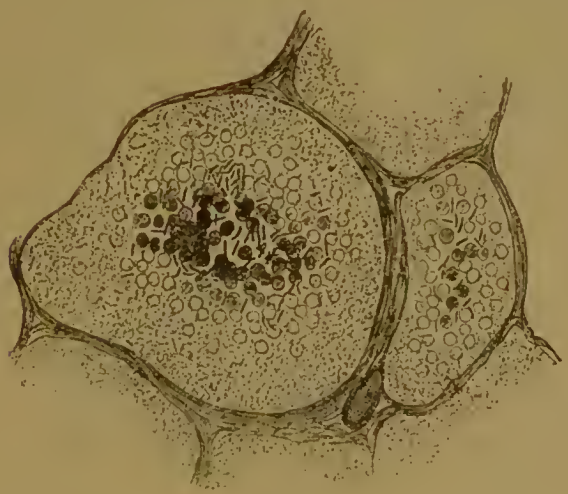


Fig. 6.

Acute miliary tuberculosis. Section of a tuberculous lung of a boy (× 350), showing two complete alveoli and four incomplete, full of organic detritus with many nuclei and the formation of lymphoid and epithelioid cells. In the centre of the two alveoli are seen several bacilli of tuberculosis. Klein.

the lungs, and whatever be the particular spot on which it alights, the injuries it produces are always the same.

The irritation produced by the slow proliferation of the bacillus of tuberculosis induces the formation of cells of three different kinds (lymphoid, epithelioid and giant) which produce a nodule of the size of a millet grain, of a greyish semi-transparent colour, to which the name of grey tubercle has been given. This tubercle gives rise to pressure on the surrounding tissues, closing the local capillary vessels, and sometimes produces a surrounding inflammation which takes the names of broncho-alveolitis, broncho-pulmonitis, endo-bronchitis, peri-bronchitis, according to the spot where the tubercle has been formed.

FIG. 7.

Portion of a lung infiltrated with gray tubercles first stage of the size of a pin's head JONES & SIEVEKING.

A characteristic feature of the tubercle is the total absence of vessels, and therefore the cells have little power of resistance and soon undergo a process of decay; they lose their

transparency, disintegrate and form a granular mass of an albuminoid nature with a little adipose substance, resembling cheese. This process of transformation has been called caseous degeneration. The tubercular nodule thus transformed and sometimes greatly increased in size has been called *yellow* or *strumous tubercle.* This caseous mass soon becomes soft and watery, penetrating into the open bronchus which communicates with the tubercle, and is then evacuated by means of the cough, thus leaving a little cavity of the size of the pre-existing tubercle which communicates directly with the atmosphere.

The injuries described, which we may call the *first stage*, are produced solely by the bacillus of tuberculosis ; but from this moment a new element is added to quicken the process of the disease and to render the injuries more serious and the process of destruction more rapid. This new element is furnished by the common pyogenic micro-organisms which are to be found in the air and which, by the act of breathing, are brought directly into the small cavity above mentioned, which was left by the expulsion of the contents of the tubercle.

A septic inflammation of the walls of this little cavity now takes place, and the result is an infiltration of the micro-organisms into the surrounding tissues, which are easily destroyed. With their destruction a quantity of organic detritus mixed

FIG. 8.

Lungs of a rabbit which had been injected with ten drops of milk taken from a cow with tuberculosis of the udder. The rabbit died after ninety-two days with general tuberculosis. These lungs are almost wholly transformed into tubercular deposits; the upper portion of the left lung is almost wholly destroyed (CROOKSHANK).

with a purulent liquid is produced ; its composition is very complex, and it is always more or less abundant in the tubercular cavities. Thus enlarged, these cavities may rapidly spread, and unite with each other, the surrounding tissue breaking down, and a real cavity may be formed varying from the size of a pea to that of an egg or an orange.

What injuries one may in the end encounter in a lung we may now imagine, for in the majority of cases tuberculosis first manifests itself at the apex of one lung, then spreads itself in such a way that the whole lung is studded with little tubercles, with the repetition in each of them of injuries such as we have described. The result is that eventually the entire lung is pierced with thousands of cavities of every size, and resembles a sponge of inferior quality in which, amid the minute cavities, larger ones may be perceived, some so huge that they may easily hold a hen's egg.

The *second stage* of the disease may be considered as the result of the presence of the septic micro-organisms in the lesions that at first were bacillary ; and the *third stage* the further injuries resulting from the breaking down of the smaller to form a great number of larger cavities, with spreading of the lesions over the whole lung. The three stages present some characteristic

distinctions tolerably well defined, but they are principally useful as explaining what follows.

The development of tuberculosis is not always as we have described ; sometimes there is a spontaneous recovery, as we often find in the post-mortem examination of patients who have died of a different malady and who offer undoubted traces of spontaneous recovery from tuberculosis from which they had perhaps suffered many years previously.

Spontaneous recovery may happen in the following ways : The irritation produced by the tubercles on the surrounding sound tissues gives rise to reactions which are not always identical. Sometimes a serous infiltration occurs in the neighbouring sound alveoli with destruction of the partitions between the alveoli and the formation of a gelatinous mass ; at other times epithelial cells are slowly formed, resulting in a desquamation of the bronchi and alveoli, this being the common tubercular broncho-pneumonia. At other times again, in the surrounding sound tissues, there is a growth of fibrinous infiltration, causing a hepatization resembling that of ordinary pneumonia. We may besides have a hard, fibrous infiltration causing the pulmonary tissue to become tough and coriaceous, forming a hard, fibro-caseous collection that remains inactive and permits us to consider the patient as recovered. This process is always a

very slow one, and goes under the name of tubercular pulmonary sclerosis.

Another and more marked method of spontaneous recovery is by the calcification of the yellow tubercles. In such cases calcareous salts settle themselves in the caseous substance of the tubercles, this substance hardening and becoming chalky, while all around it there is a formation of connective tissue which thus constitutes a capsule; by means of the latter the tubercular substance is completely secluded and remains harmless for the whole life. Such a calcareous substance, however, loses nothing of its virulence, and an injection made from it into an animal would be followed by tuberculosis, probably because the "spores" of the bacilli may remain for an indefinite time shut up in the calcified tubercle without losing their vitality.

We must finally remark that by good and scientific nutrition and respiration, of which more hereafter, the healing of some cavities may also be brought about; the lesions contract and cicatrise by the formation of connective tissue, and the latter finally shrinks, changing the cavity into a nodule of cicatricial tissue. These spontaneous recoveries, unfortunately, are very rare, only comprehending those cases of tuberculosis in which the foci of injuries are very few. When these are numerous a few may spontaneously heal, whilst the others follow their course and spread the disease.

CHAPTER IV.

HOW THE BACILLUS ENTERS THE LUNGS.

The bacillus of tuberculosis may enter the lungs from the external air, with which it is introduced into the bronchi, or it may be carried there from some part of the body in which it previously existed.

We frequently observe that an individual who had been previously affected with tabes mesenterica is attacked with tuberculosis of the lungs before his death, owing to the passage of bacilli from the abdominal organs to the lungs. Sometimes we see scrofulous children with enlarged cervical glands, who are attacked by rapidly fatal diffused tuberculosis of the lungs. In this case the bacilli of tuberculosis, following the route of the lymphatics, entered the thoracic duct and thence were carried by the blood to the lungs, forming very numerous foci of disease in both organs at the same time (see *Fig.* 7, page 16).

In many cases of tuberculosis of the lungs one learns that two or three years previously, or even

more, the patients were ill of pleurisy. This pleurisy was evidently of a tubercular nature; and very probably the germs of tuberculosis invaded the lungs through those lymphatics, which, originating in the pleura, afterwards enter the lungs.

In general we may state that a tubercular focus existing in any part of the body constitutes a perpetual danger of the bacilli being carried to the lungs; though we must own that this occurrence is not universal. For instance, in lupus the passage of its bacilli to the lungs is rare, while a tubercular anal fistula is almost invariably followed by tuberculosis of the lungs.

But by far the commonest way for the germs of tuberculosis to enter the lungs is by respiration. We have already stated what an extraordinary quantity of bacilli a consumptive person may emit in twenty-four hours only by expectoration. He emits perhaps a greater quantity by fits of coughing, even without spitting, by sneezing, by speaking, etc. This is proved by the highly important experiments of Flügge, who showed that a great quantity of such germs is to be found in the little drops of "spray" emitted by the consumptive person in such movements. So that the room of the patient is highly tainted by an almost inconceivable number of micro-organisms of tuberculosis which alight on the floor, the furniture, the

walls, the bed-clothes, the chair coverings, etc., and float continually in the air of the room.

This must be the necessary conclusion when one remembers that the expectorations dry and change into dust, easily lifted by the movement of the air in a room ; that the micro-organisms contained in the minute drops of water emitted by coughing, sneezing, etc., are already in a state that renders it easy for them to remain suspended in the ambient air ; one must bear in mind besides, the extreme smallness of the bacilli of tuberculosis, which have a length that varies from 2 to 4 micro-millimetres, that is to say, from two to four thousandths of a millimetre.

It is true that latterly so much has been said and repeated about the public danger that attends the dust of dried sputa that, at least amongst better educated families, care is taken not to expectorate on the floor, and the sputum is carefully disinfected ; but nothing has been done as yet to counteract the micro-organisms ejected with the minute drops of water in breathing, and therefore we may be sure that the rooms where consumptives live always contain a great quantity of bacilli.

We may, moreover, be sure that those who live in or visit the house of a consumptive patient breathe tubercular bacilli in abundance. In the Hospital of la Charité at Paris, Professor Strauss experimented on the subject. He examined the

nasal contents of twenty-nine persons belonging to the hospital (assistants and nurses of both sexes) by putting in their noses little balls of cotton-wool to gather the dust they inhaled. On nine of them he found the bacilli of tuberculosis. This means that, if in a single experiment nine out of twenty-nine were found to have inhaled bacilli, repeating the trial at greater length would result in the bacilli being found in all. We therefore do not hesitate to conclude that no one escapes inhaling the bacilli of tuberculosis at some period of his life ; and we should all be liable to die of tuberculosis if our bodies did not offer very powerful natural means of defence against this terrible enemy.

We frequently see the first case of tuberculosis in a family followed by a second, then by a third, and so on, until sometimes the whole family is destroyed ; but it is also true that sometimes we see one case only.

In order to explain these differences many medical men suppose the existence of a peculiar *predisposition* to consumption. I do not mean either to deny or admit this contention ; I only wish to state that I fear this expression, for its acceptance puts a stop to further investigation that might throw light on some facts which are now too easily explained by this word *predisposition*, which, after all, no one can define either as to its nature or extent.

I have lately had the opportunity of making observations in a family composed of eleven children, all very healthy and strong. Ten years ago one of them came home from Massowah where he had served as a soldier, ill with consumption of the lungs. Not long after he died, and within ten years five of his brothers died. Of the other five still living three are consumptive, one of whom, a lady, once stout and strong, is now in an advanced stage of illness. Up to the death of the first who was consumptive, tuberculosis had been unknown in that family, and certainly the notion of speaking of predisposition in this case, as in many others, means simply an endeavour to conceal by a word certain facts, the cause of which escapes our penetration.

I may be mistaken, but I am convinced that what is called predisposition is principally a low condition of health in those who fear to catch tuberculosis, produced by a number of precautions which bring about precisely the opposite effect to that intended, that is to say, paralyze the efficacy of those natural means of defence that we, all of us, possess.

As already mentioned, our air passages, beginning with the nose, are lined with a layer of epithelial cells which, until the alveolar passages are reached, are furnished with vibratile cilia. They are kept moist by a considerable quantity

of mucus, and therefore any foreign substance that may penetrate through the air canals is soon stopped by the viscosity of this mucus and eliminated by the movement of the vibratile cilia.

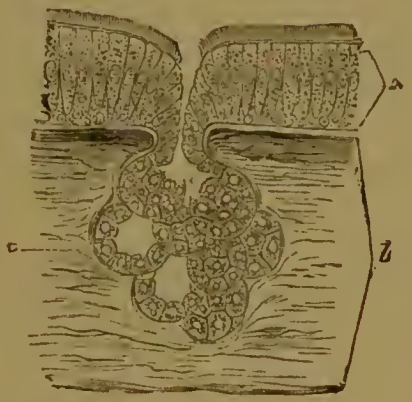


FIG. 9.

Section of the mucous membrane of the trachea : (*a*,) Epithelium that lines the mucous gland with its vibratile cilia ; (*b*,) Strata of which the mucous membrane is formed ; (*c*,) Mucous gland.

The particles of dust are in great part arrested by the mucous membrane of the nose, the trachea and the large bronchi. The minutest parts of foreign substances, micro-organisms included, if they succeed in passing these first obstacles, must necessarily fall on the sides of the medium-sized bronchi, where, too, they are stopped and removed. Should it happen, on account of their considerable quantity, that some are successful in passing the middle bronchi, their progress will be arrested in the minor bronchi, which, as we have already seen open and shut automatically at every inspiration

and expiration; then they are eliminated in the same way. Therefore we may conclude that when the breathing organs work normally the peril of contracting tuberculosis is very small. This is proved by the fact that, though the micro-organisms of tuberculosis are so abundant, nay, though some individuals live many hours of the day in rooms where such micro-organisms are in enormous quantity, they yet escape infection. The danger becomes really great, however, when the function of this epithelium is impaired in consequence of some local congestion, or the function is thoroughly suppressed for a time, as in the case of a bronchial inflammation. The inflammation produces the detachment of the epithelial cells, causing a real excoriation.

Now, to produce a state of congestion in the bronchial mucous membrane and thus to predispose to common bronchitis, nothing is more favourable than the precautions which were suggested even by physicians in bygone times, and which still find great favour with the lower classes. Such precautions are generally adopted by those who have the greatest fears of contracting tuberculosis, that is to say, by those who have had some consumptive person in the house, by those who are in weak health or whose thorax is deformed. Such persons are quite afraid of pure air. In their houses the windows are constantly shut in winter,

or they have double glass frames, or sometimes the windows are lined with paper or cloth strips ; woe to them if a door be opened, or worse, if a draught of fresh air be let in !

What kind of air exists in such apartments it is easy to imagine ; great quantities of organic particles continually settle on the walls, furniture, etc., so that rooms and furniture acquire a peculiar smell. In this way a splendid medium is formed for the development of an extraordinary variety of micro-organisms, which, in union with the gas of putrefaction of organic matter, with the minutest particles of the organisms themselves, form the elements that are continually introduced into the bronchi. Thus, by means of breathing, the material fittest to produce a state of chronic congestion of the bronchial mucous membrane is introduced, and this condition, while it impairs the activity of the epithelium, predisposes to bronchitis. As if all this were not sufficient, the people who live in such apartments lead the same life out of doors. In offices they live with shut windows, sometimes in the smoke of tobacco, so that they create the fittest atmosphere for bronchial irritations and organic depression ; in workshops ventilation is always carefully avoided. Children or babies are shut up with every precaution, in rooms where the air cannot even penetrate through chinks, and they are taken out

to walk only when a perfect calm in the atmosphere reassures the parents against any peril from a draught. The injuries resulting from this fear of fresh air are not limited merely to the bronchial mucous membrane. They impair all the forces through the imperfect oxygenation of the blood and the introduction into it of many volatile hurtful products, which strike directly at the cells by which the body is built up and which work together to maintain life. Such persons lead a sickly life, physically and morally listless, and this constitutes a real predisposition to any kind of illness.

If one of these individuals is exposed to the bacillus of tuberculosis we may be sure that he will easily contract the disease, for the bacillus will settle on a bronchial mucous membrane already torpid, deprived of every capacity to drive it away, nay, forming a suitable culture bed for its growth. This is the real predisposition we all must bear in mind, and avoid at all costs.

There is a physiological fact which, while it supports this truth, shows the great importance that sedentary life in shut-up rooms has in promoting this disease. We have seen in the chapter discussing breathing that the pulmonary apices advance for the breadth of three fingers above the collar-bone on both sides of the neck. The collar-bone is motionless and does not rise and fall in breathing, and the result is that by

ordinary and calm breathings the pulmonary apices have almost nothing to do. The air penetrates them only in great inspirations. Hence, these slight bronchi, which we have seen are deprived of cartilage, opening only in inspiration and expiration, are almost always inactive at the apices. Now, if the bacillus of tuberculosis introduces itself into these apices, it will be expelled with much more difficulty, for if by a strong inspiration it has been carried into a small bronchus, it will stop there because this tiny ramification will not open again perhaps during many hours; that is to say, when the bacillus has already produced a certain degree of irritation, increased the local secretion, and at the same time blunted the activity of the epithelium. This becomes the starting point of tuberculosis. In fact, out of a hundred cases of pulmonary tuberculosis, certainly ninety-five, and perhaps more, will show the first sign of disease at one of the pulmonary apices. Very seldom do we see the malady begin in other portions of the lung; and even when it is so, if we examine attentively, we see sometimes that in reality the origin was at the apex but it diffused itself to another region in the manner we shall mention later.

This important and generally acknowledged fact shows of what importance a full, large and unimpeded breathing has in the prevention of

tuberculosis, and, on the other hand, how easily those may contract it who, from well grounded fear of the disease, adopt what seem to them preventive measures, as, for instance, the morbid avoidance of fresh air as of their worst enemy, while in reality it is their only safety.

Another and kindred fact should be widely known because it bears upon what has been already said, and may assist in abolishing a common and most injurious custom. We have seen that the apex of the lungs expands well only in the case of full inspirations. Consequently, with women who habitually tighten their corsets the pulmonary apices are practically inert, and the danger of attack from tuberculosis is appreciably greater. The corset compresses principally the lowest ribs, that is, those having the largest range of expansion under the deepest inspirations. These inspirations, so favourable to the purification of the apex of the lungs, are very incomplete under these conditions; hence the greater liability to pulmonary tuberculosis. This is why in Italy consumption is much more prevalent among women of a certain age than men, and why the discrepancy only covers the period during which the effects of the corset are most felt. The following figures give the mortality from consumption in Italy during the year 1897, classified in regard to sex and age.

DEATHS BY CONSUMPTION IN ITALY IN THE YEAR 1897.

AGE	MALES	FEMALES
From 0 to 5 years	1,144	1,122
,, 5 to 10 ,, ...	455	677
,, 10 to 15 ,, ...	444	1,150
,, 15 to 25 ,, ...	4,598	6,297
,, 25 to 35 ,, ...	4,235	5,210
,, 35 to 45 ,, ...	2,656	3,191
,, 45 to 50 ,, ...	1,062	1,094
,, 50 years upwards	2,981	2,418
Total	17,581	21,089

We here see that during the first five years of life the mortality in males and females is the same; after that the use of the corset begins, and we soon observe (from 5 to 10 years) a considerable increase in the death rate amongst females* ; this continues to be very great from 15 to 25 years, lessens from 25 to 39, and continues to decrease until between the ages of 45 and 50 it disappears. Later than 50, that is, after the corset age is passed, the mortality is greater amongst males, as it probably would be throughout were it not for the corset. Men are doubtless much more liable to tuberculosis than women, for they live more in manufactories, mills and workrooms where, besides living in vitiated air caused by

* In many parts of Italy, as for instance, in the Marches and in Umbria, in the Abruzzo, etc., the corset begins to be worn at a very early age.

the crowding of many persons in one building, they are in other conditions very favourable for contracting the disease. This is proved by the following figures which represent the mortality due to pulmonary tuberculosis for the same year (1897) in England. In that country factories are much more numerous than in Italy, and men are consequently more in peril of tuberculosis. Moreover, corsets are less worn than with us, so that English women run less risk of falling a prey to consumption.

DEATHS BY CONSUMPTION IN ENGLAND IN 1897:

AGE	MALES	FEMALES
From 0 to 5 years	797	623
,, 5 to 10 ,, ...	383	345
,, 10 to 15 ,, ...	716	3,955
,, 15 to 20 ,, ...	5,332	4,335
,, 25 to 35 ,, ...	5,322	4,645
,, 35 to 45 ,, ...	5,325	3,900
,, 45 years upwards	7,033	3,943
Total	23,046	18,596

We notice that between the ages of 5 and 15, the period during which the influence of factories is not felt, the mortality amongst females in England is greater than amongst males. The slight difference which is observed in the first five years of life appears to be accidental; for instance, in the year 1895 there were during this period 772 males and 736 females.

The proportion in figures thus stated is constant in the two countries. Did space and time permit, I could explain why the mortality from consumption is very much heavier in Italy during the early periods of life than in England, and why these conditions are reversed in old age. This demonstration would serve to prove still further the great truth that the more respiration is interfered with and the air is vitiated, the greater is the prevalence of this dread disease.

In conclusion, we should bear in mind the following facts: (1,) The anatomical constitution of the bronchiole and the function of its epithelium; (2,) The fact that the bacilli of tuberculosis are very numerous, and are inhaled by all at some period of their lives; (3,) Notwithstanding this, and though living in continual contact with patients suffering from the disease, many persons remain perfectly free from it; (4,) Tuberculosis almost always begins at the apices of the lungs, where, from their anatomical situation, the breathing is very weak and where the epithelial stratum possesses little activity; (5,) Those who inhale a vitiated atmosphere, such as might be found in prisons, boarding schools, eating houses, ill-ventilated workshops, etc., are more liable to succumb to the malady, because the foul air produces a chronic bronchial congestion by which the functional power of the epithelium is diminished; (6,)

Every hindrance to free breathing increases the peril of tuberculosis by lessening the respiratory function of the apices of the lungs, as is shewn by the influence of corsets in women.

Thus we may be satisfied that though the respiratory tract has always shown itself by far the most prone to cause pulmonary tuberculosis, yet the peril becomes trifling even to those who are most exposed to the infection if they will arrange to inhale pure air, as far as possible, and to maintain such full and free breathing as will suffice to maintain the bronchial epithelium in a sound and active condition.

CHAPTER V.

HOW THE BACILLUS SPREADS IN THE LUNGS.

BEFORE we can solve this problem we must recall some facts concerning the behaviour of the bacillus of tuberculosis in other tissues.

In that form of tuberculosis of the skin known as lupus, we see that the bacillus does not encroach rapidly. The disease generally runs a long course—fifteen, twenty, or even thirty years—and when it is possible to kill the bacillus, by a deep and wide-spread cauterization, the recovery is complete without relapse. The bacillus of lupus has been very rarely known to transfer itself to other organs, as for instance to the lungs, there to produce tuberculosis. In tubercular enlargement of glands we observe an identically chronic course, and not infrequently after a very slow suppuration lasting many years, recovery takes place and the patient lives to old age in perfect health. In these cases the spread of the bacillus from the glands to the lungs is by no means rare, but the facts nevertheless show that such secondary infections form

the exception. Precisely the same characteristics are exhibited in tubercular caries of the bones. And here too recovery is perfect without the spread of infection, when the surgeon has wholly excised the portion of affected bone and completely disinfected the wound.

In tubercular arthritis, the so-called fungous arthritis, the same observations may be made. It is common for a patient whose arm, foot, or leg, has been amputated for this disease, to enjoy perfect health and live to a green old age. The course of tuberculosis is widely different when the specific bacillus attacks a serous tissue, such as the peritoneum, the meninges, or the pleura. In such cases it spreads much more swiftly and produces diffuse peritonitis, meningitis, or pleuritis, which is followed sometimes by rapid death.

The greater virulence of the bacillus when attacking the serous membranes may be due to the facility with which the bacillus is carried to other parts of the membrane: (1,) By the continual movement of these serous tissues; and (2,) By the liquid which moistens their surfaces, and which forms a thin fluid stratum between the two sheets. It is very probable that the bacillus of tuberculosis when it settles between the two layers of the pleura, by multiplying there is carried over the entire surfaces of both by their continual motion. A similar course is

probably followed in the case of other serous membranes.

These facts prove that the bacillus of tuberculosis has, of itself, little tendency to spread, and that when this takes place rapidly it is due, not to movement of the bacillus itself, but to some agency by means of which it is mechanically carried.

After these observations let us examine a simple case. A young lady, strong and healthy, who had never been ill, with healthy parents, and having no history whatever of tuberculosis, married a young man whose father and brother had died of consumption. After some years of married life symptoms of tuberculosis appeared in the husband, and he died seven years later (1895), leaving her a widow with two children. She left the neighbourhood and went to live in a small town with her children, hired a large, sunny, well-aired dwelling, and spent the summer in the country. From 1895 she had good health ; but last June she began to feel a general fatigue, a weakening of her strength, her appetite failed, she lost flesh, and had some attacks of dry cough without expectoration. I examined her at the beginning of last October and found an increase of the vocal thrill at the apex of the right lung, hypophonesis, a rough breathing, but neither râle nor rhonchus. The remaining part of the right lung was sound, the left one quite sound.

It is a common occurrence for an individual who has nursed a consumptive patient to show symptoms of the disease at the distance of two, three, four, or ten years, even when the person stricken has left the neighbourhood directly after the death of the first patient; and I believe this fact alone authorizes us to conclude that the person so stricken contracted the illness when nursing the first patient. It remains now for us to explain how an infection of pulmonary tuberculosis could remain latent so many years without producing the slightest symptoms, or at least, such trifling phenomena as a short dry cough, a liability to colds, etc., that the new patient scarcely heeded them.

Many of the diseases attacking the human organization being abrupt in their mode of invasion, it has become a habit to date the onset of a malady from the period when the sufferings of the patient commence. This is a somewhat serious mistake, for the majority of chronic diseases do not cause any trouble at the outset. Interstitial nephritis, cirrhosis of the liver, some tumours, etc., do not give any trouble in their earlier stages; and when suffering compels the patient to have recourse to the physician the work of destruction is already considerable. I have seen lately a primary cancer of the liver which had never made itself felt, so much so, that the patient had recourse to the

physician, not because of the cancerous liver, but because he had noticed a hardening at the right breast, the original disease being only discovered through this secondary manifestation.

So it is with pulmonary tuberculosis. When we have convinced ourselves that a person after having caught the disease may live many years before he is aware of it, we must conclude that the progress of pulmonary tuberculosis is very slow in the more latent and initial stages. It needs little reflection to see that, generally, this cannot be otherwise. We have already shown how difficult it is for the bacilli of tuberculosis to settle in the lungs, principally on account of the obstacles set up by the bronchial epithelium. The consequence is that an extensive pulmonary invasion can never take place through the respiratory tract, unless from some morbid condition the bronchial epithelium does not work. In the great majority of cases there never has been such a sudden general invasion, but a few germs settle, principally at a pulmonary apex, and there form one or two foci. Probably, too, some of these single bacilli are carried by the lymphatic current to a neighbouring gland where an irritation and a subsequent enlargement take place, which may last for years without giving trouble, precisely as we see daily in the glands of the lower jaws and those of the neck. Or it may happen that these few germs settle in the

pulmonary tissues and give rise to the formation of a tubercle, this tubercle developing itself and following the course previously described. From the little tendency that the bacillus has to spread, such a tubercle may remain the only one for a long time, or at most, give rise to a new focus in an alveolus or infundibulum close by.

When the softening and breaking up of the contents of the tubercle have taken place through an attack of coughing, the peril of diffusion increases, for the germs of tuberculosis may now settle in sound tissues principally in two ways. By the process of inspiration the tubercular substance itself, which is very effective for mischief, may be driven into a ramification of the bronchus or a healthy aveolar canal, and there form a new focus. Or the bacilli of tuberculosis themselves, which by the cough were driven into the minor bronchi, may in the same manner by inspiration be transplanted to form new foci of disease in sound places.

It is highly probable that this second method of auto-infection is the most frequent, on account of the greater facility with which the bacilli are transplanted by the action of inspiration.

Here, too, we must remark that this process of diffusion will be the more difficult and slow in proportion to the condition of health of the bronchial epithelium which will oppose these auto-infections, precisely as we have seen it oppose itself

to infection from without. Therefore we must again insist on the paramount importance of respiration in a free and pure air without hindrance to the breathing movements, in preventing auto-infection, that is to say, the diffusion of the malady to the whole lung.

In this early stage it is evident that the patient does not suffer any disturbance of his general condition; the presence of one or of a few tubercles as big as a grain of millet cannot disturb the state of health, nor could a physician discover anything wrong. This condition may last some time, and even terminate by calcification or cicatrization, as we have seen. But when through auto-infection new foci have been formed, evidently the danger of diffusion is doubled, or trebled, so that the process of auto-infection will become the more rapid, the greater the number of foci. This we continually see. When we observe infiltration at the apex of a lung, we generally see it followed by a rapid invasion and by the disease of the remainder of the lung, the rapidity being due to the great number of tubercles that the patient already presents when he consults the physician; usually, too, the diffusion quickly spreads to the other lung. The consecutive invasion of the second lung is a constant fact, failing the arrest of the disease in the first; and remembering the anatomical relations existing between the two, we

must necessarily conclude that the extension can only be explained in the manner described.

Flügge by his important experiments has proved that a consumptive patient emits a great quantity of bacilli in the minute drops of water from his mouth and nostrils when he coughs, sneezes, or speaks loudly, because the air passages hold in abundance the bacilli of tuberculosis. Hence the unavoidable spread of the disease by auto-infection during inspiration ; this is greatly increased after a tubercular infiltration has taken place in a part of the lung, the tubercles being then so numerous as to produce an inflammation of the surrounding tissue, against which the defence of the bronchial epithelium is reduced to nothing. The bronchial epithelium screens the lungs from the action of external agents, which are introduced by inspiration, precisely as the horny superficial layer of the skin defends our organism from the action of atmospheric agents. This action is manifested immediately after the skin has been injured, and the same thing occurs in the bronchi.

We therefore infer that the course of pulmonary tuberculosis from the first depends on the different agents we have mentioned, which enables us to understand why some cases spread rapidly, while others only give the first symptoms of their existence after the lapse of several years.

It is now easy to explain facts which are commonly attributed to *individual predisposition*, *feeble organic resistance*, *habitus phthisicus*, etc. Very frequently, for instance, one observes a pulmonary tuberculosis develop after typhoid fever. In typhoid the patient must lie in bed from forty to fifty days, and even longer, during which time pulmonary stasis takes place. The protection of the epithelium consequently fails, and tuberculosis appears, especially if some separate and unobserved tubercles already existed. Tuberculosis in consequence of influenza, scarlet fever, small-pox, and generally any long enfeebling malady, is explained in the same way, without having recourse to such obscure, inconclusive and unintelligible expressions as *inferior resistance*, *predisposition*, *tuberculotic habit*, etc.; words which almost seem created to satisfy minds that naturally wish to know the hidden reasons of observed facts.

CHAPTER VI.

THE THREE STAGES OF TUBERCULOSIS AND THE POISONOUS PRINCIPLES.

We have already divided the course of tuberculosis into three stages. The first begins from the moment in which the bacillus settles in the pulmonary tissue, and, as before remarked, the commencement of this stage escapes the patient's observation entirely, and therefore the physician is not called in. The second stage begins with the action of the common atmospheric pyogenous germs, which are carried by inspiration to the little wounds caused by the emission of the contents of the tubercles in a paroxysm of coughing. The third stage, according to our definition, is the result of the injuries that have been produced during the first two.

It is evident, from what has been said about the course of tuberculosis, that it is practically impossible to observe an injured lung, in only one stage of the disease. It is often possible to find simply the first, but it is common to meet

with large cavities belonging to the third stage, while in other spots in the same lung tubercles of the first stage, and little injuries of the second are to be found. This classification nevertheless has its practical value in determining the nature of the injuries, the course of the illness, and the prognosis; and also in showing the importance of some of the destructive principles which are formed in the lesions and absorbed by the blood, and by it brought into the system, poisoning the cells of the most vital organs, and becoming the real cause of the patient's death.

These poisonous principles, which may be all comprehended in two great categories—ptomaines and toxalbumins, are due to different invading micro-organisms, and differ according to their variety and quantity, and also according to the stage of tuberculosis. The injuries of the first stage being due solely to the bacillus of tuberculosis will contain only the poisons that such a bacillus is able to elaborate. These products are not yet well known. Koch's tuberculin is a glycerine extract of the produce of the tubercle bacillus in pure cultures. He has found that this substance produces a considerable reaction, local as well as general, in patients affected with tuberculosis, especially in the cutaneous form, lupus. We all remember the extravagant joy that arose, when it was believed that by this tuberculin

a sure specific against tuberculosis had been discovered. Nowadays tuberculin is chiefly employed to detect tuberculosis in a suspected animal, especially a cow, when ordinary means have failed. The tuberculous cow reacts to an injection of tuberculin, with a fever more or less intense, while the sound animal has none.

Soon after, Professors Crookshank and Herroun obtained from the cultures of the bacillus a ptomaine and an albumose. They observed that while the ptomaine produces an increase of temperature in tuberculotic animals with a marked enlargement of the tubercular glands, in sound animals a lowering of the temperature was observed. The albumose too produces a heightened temperature in tuberculotic animals, but instead of causing an enlargement of the tubercular glands it makes them more definite, hard and painful.

In March, 1897, Koch announced the discovery of three new preparations. Tuberculin TA, tuberculin TO, and tuberculin TR. Behring too discovered a new tuberculin TDr.

It is not known whether the tubercle bacilli elaborate in the lungs the same products which are cultivated in the laboratory. At any rate it is almost certain that these principles are not those that produce the more serious phenomena of the blood poisoning, as will be shown later.

During the second stage the conditions change wholly. The purely tubercular injuries of the first stage being now brought into communication with atmospheric air, are contaminated by numerous septic micro-organisms which exist in the air, the common pyogenic germs; and we may imagine what then occurs by recalling the state of our surgical wards before the introduction of anti-septic surgery. Then, blood-poisoning due to the infection of wounds by micro-organisms was common, and assumed all degrees of gravity, being variously named, septicæmia, pyæmia, or nosocomial or hospital gangrene, according to the different forms presented.

Almost all these forms of poisoning may start in the same way. Cold shivers followed by fever give place to abundant sweating, with abatement of temperature, prostration, loss of appetite, etc.; then cold shivers again, with repetition of the same phenomena, and death after a variable number of these paroxysms according to the quantity and quality of the substances absorbed in the blood.

In the second stage of tuberculosis we have a variable quantity of small lesions, which becoming septic, reproduce exactly this process in all its forms of intensity, according to the number, the extent, and the degree of septicity of the injuries.

Beginning with the slight tremors, sometimes simple horripilations, one may reach great daily

rigors, and the fever resulting from them may vary proportionately in severity. Now it is a heightened temperature scarcely perceptible ; only two-, three-, four- tenths of a degree, not more ; but however trifling, as it is repeated day by day, generally towards evening, we must be able to give it its true meaning. From this light degree we may see the whole range run through until we have a daily temperature of 104°, 105°, and sometimes even 106°, these temperatures being in strict proportion tot he quantity and quality of the poisonous principles that have been formed in the lung injuries and have been absorbed by the blood. The ensuing perspiration which generally takes place in the early morning assumes also all gradations, from a slight to an abundant sweat that wets the shirt through. The prostration that follows the lessened appetite and emaciation will vary in strict relation with the phenomena just described, so that some cases terminate rapidly in death, while others move with the greatest slowness. All this reproduces faithfully the picture of blood-poisoning by septic wounds.

And just as in our surgical wards, before the antiseptic treatment, the greatest danger lay in the infection of wounds, so it is in tuberculosis. Death is not caused by the number of lung injuries, or their extent, but solely by septic infection of these injuries. On this point all authors agree,

and Professor Foà teaches "that consumptive people do not die so much because the lung is destroyed by cavities, for a little of sound lung is sufficient to continue the function though vast parts are destroyed; and of this we are often convinced when we see consumptive lungs that preserve so trifling a zone of functioning viscus, that we wonder how the patient could keep alive so long; but they die principally from septicæmia or sapræmia, that is to say, from infections that pervade their organism."

In some cases consumptive patients succumb with lung injuries of little extent, only because in such injuries very virulent septic infections have taken place, and have produced septicæmia or sapræmia; at other times the infections are less violent, and the patient can live until a slow and total destruction of the lungs has taken place. Just as, before the days of antiseptic surgery, individuals who had been submitted to very slight operations sometimes died, so it happens with lung injuries which sometimes fall a prey to such virulent infection, being put in the same direct communication with atmospheric air that external wounds were, that the sick person dies without any considerable lesion of the lung at all.

That such phenomena of infection are produced only in the second stage and not in the first, that is to say, are caused by the presence of the

common pyogenic micro-organisms of the air, is also proved by many facts which fall under our observation, such as the following: A lady of thirty-five years, dwelling in a large city, on account of a large tubercular injury reaching from the apex of the left lung to the fourth rib, was a prey to daily high fevers, which rose to 104°, with abundant sweating, emaciation, etc. She was sent by her physician to a sanatorium placed on the mountains of Switzerland. After a fortnight's stay the fever ceased altogether, so that she could benefit by the overfeeding adopted in sanatoriums in general; she grew stouter and increased much in weight, for the first time in her life. When she returned to town after eight months, although the lung injuries were not improved, the general conditions had undergone an immense change for the better. After a few months' stay in town the fever reappeared, accompanied by all the other phenomena of ill health. A second visit to the sanatorium produced the same benefits as the first, and after a six months' stay she did not return to town, but settled in a country house situated in one of the lower Piedmontese Alps, where the fever did not reappear, though the lung injuries did not heal. Nay, even in the sanatorium they continued to spread, so that the tubercular infection has now occupied the entire left lung and the apex of the right. Notwithstanding this considerable injury

the lady has no fever, can eat moderately, and maintains the weight she acquired at the sanatorium, a weight she never had before even in good health.

We know, and it has been often proved by analyses that have been made of the air of towns, the country, the seaside, and of mountains at different heights, that while town air is extremely rich in micro-organisms, the mountain atmosphere is almost free, and at a certain height contains none at all. A consumptive person who leaves his city dwelling for the mountains where sanatoria are generally placed, leaves an atmosphere fouled by micro-organisms that cause septic infections of his lung injuries, to breathe an air where these are absent; in other words, where the causes of fever, night-sweats, etc., do not exist. This is the true explanation of the cessation of these infective phenomena; moreover, as the specific bacillary injuries do not disappear at all, we must necessarily conclude that the toxins elaborated by the bacillus of tuberculosis are not the cause of the severe toxæmic phenomena that tuberculous patients exhibit, but that the latter are exclusively due to the action of the atmospheric pyogenic micro-organisms, and such phenomena must be the more fatal the longer the patient breathes an air abounding with them, and *vice versâ*.

Nor is this all. The septic infections which characterize the second stage of the pulmonary lesions do not limit themselves to general phenomena. On the surface of the lesion itself they produce a septic inflammation which rapidly destroys the tissues; the small cavity left by the expulsion of the contents of a tubercle grows larger, coalesces with neighbouring cavities in which the same process is occurring, and thus hastens a breaking down of the lung which otherwise would have been very slow. With this rapid work of destruction we reach the—

Third stage. While the two preceding stages differ more particularly in character, the third differs from the second rather in the extent of the lesions. Here the cavities are numerous, and may be counted, not by hundreds, but by thousands. The greater number have the size of a grain of corn, those as big as a hazel nut are numerous, here and there some are as large as a walnut, and generally at the apex a few are observed of the size of a hen's egg, or even larger.

Owing to this condition of the lung, it may happen that a portion of it has no communication with the atmosphere. The bronchi that should reach certain localities of the lungs find the path obstructed by numerous lesions, which, deprived of communication with the outside may be considered so many septic foci, where the

process of destruction moves apace by reason of the activity of the germs they contain. These cavities contain a greyish, purulent liquid, full of organic detritus, so that the section of a lung in this stage presents the most pitiable of sights, and is a good demonstration of the degree of destruction that can be produced by the combined action of the bacilli of tuberculosis and the atmospheric micro-organisms found in the great centres of population.

CHAPTER VII.

TRANSMISSION OF TUBERCULOSIS BY HEREDITY.

LET us briefly examine the question of heredity as one of the means by which the bacillus of tuberculosis invades our organization. To-day the general trend of medical opinion is to deny inheritance of tuberculosis, but to contend for an inherited *tendency* to the disease, a predisposition, a tuberculotic habit, etc.; so that all who are born with this predisposition are candidates for infection (this is the favourite expression of the moment).

The principal grounds for the support of this theory are the following: The bacillus of tuberculosis circulates only with difficulty in the blood; when this occurs it is followed by an acute miliary tuberculosis that in a short time kills the individual. Therefore, a tuberculous mother cannot easily transmit the bacillus to the *fœtus in utero*; and if any bacilli exist in the blood, they have to pass through the filter of the placenta before reaching the fœtus. With greater difficulty still, it is added, can a tuberculous father, through sexual intercourse, so communicate the bacillus that the fœtus in formation may contain and develop it. Let us

examine these facts. Very frequently we see primary fungous arthritis. In this case how, if not through the blood, did the bacillus reach the injured articulation? Again, how did the bacillus invade a testicle in *primary* tuberculosis of that organ? Whence did it come in *primary* tubercular osteitis? How, also, does it reach the brain, there to form a solitary tubercle? In these and in many similar cases one must necessarily admit that a bacillus of tuberculosis has found its way to the blood and been carried to the place where it has settled and multiplied.

A number of tubercular fœtuses have also been found in the uterus, proving unequivocally that tuberculosis may be inherited. But not only so; the inherited nature of the malady is shown by the following figures:—

DEATHS FROM TUBERCULOSIS IN ALL FORMS THROUGHOUT ITALY DURING THE YEAR 1897.

PERIOD OF LIFE.	TOTAL DEATHS.	MONTHLY AVERAGE.
During 1st month	202	202
,, other 11 months	3,405	309
From 1 to 2 years	3,771	314
,, 2 to 3 ,, ...	2,003	177
,, 3 to 4 ,, ...	1,185	99
,, 4 to 5 ,, ...	913	76
,, 5 to 10 ,, ...	3,136	52
,, 10 to 15 ,, ...	2,653	44
,, 15 to 20 ,, ...	5,484	91
,, 20 to 30 ,, ...	12,719	106
,, 30 to 40 ,, ...	8,333	69
,, 40 to 50 ,, ...	5,313	44
Upwards of 50 ,, ...	6,676	—

We see by this table that the rate of mortality is highest during the first and second year of life, and that the death rate is also very great in the first month of life. Bearing in mind the slow progress tuberculosis makes, we can but attribute the high mortality in the two first years, and especially in the first month, to infection by the mother. My explanation of the figures is this: the high death rate in the first years of life is due to maternal infection ; later this influence lessens, while that of external causes has not yet had sufficient time to make itself seriously felt. This is the reason for the low rate shown from the fifth to the twelfth year or thereabouts of life. After this age the effect of external causes is in full activity, while the influence of heredity is reduced to almost nothing, and thus we have again a high mortality which is, however, below that of the two first years of life.

In order to appreciate fully the monthly average of the death rate after the thirtieth year, we must remember that it is lower than that of infants, because the number of individuals diminishes with advance of age. To obtain correctly the mortality by tuberculosis in different ages we should know the proportion of deaths per thousand of the population of the same age. I made this calculation on the deaths during 1895, and the results obtained were the following:—

PER THOUSAND OF POPULATION :—

From	0	to	1	year	there	died	3·9
,,	1	to	2	years	,,	,,	4·7
,,	2	to	3	,,	,,	,,	2·7
,,	3	to	4	,,	,,	,,	1·5
,,	4	to	5	,,	,,	,,	1·2
,,	5	to	10	,,	,,	,,	0·96
,,	10	to	15	,,	,,	,,	0·90
,,	15	to	20	,,	,,	,,	1·3
,,	20	to	25	,,	,,	,,	2·9
,,	25	to	30	,,	,,	,,	2·6
,,	30	to	35	,,	,,	,,	2·3
,,	35	to	40	,,	,,	,,	2·0
,,	40	to	45	,,	,,	,,	1·8
,,	45	to	50	,,	,,	,,	1·5
,,	50	to	55	,,	,,	,,	1·5
,,	55	to	60	,,	,,	,,	1·3
,,	60	to	65	,,	,,	,,	1·4
,,	65	to	70	,,	,,	,,	1·2
,,	70	upwards			,,	,,	1·3

The above figures, which illustrate the susceptibility to tuberculosis at various ages, compel us also to admit the influence of heredity, for in no other way can we explain why in children a few months old, who cannot yet have felt the effect of external causes of tuberculosis, the mortality is so much higher than in adults who have, it may be, for twenty or thirty years been exposed to these influences.

It seems, therefore, right to conclude that tuberculosis *is hereditary*, but the influence of inheritance is especially felt in early years and lessens slowly until it becomes almost *nil* after the tenth year of life.

CHAPTER VIII.

DIAGNOSIS.

WE need not discuss the diagnosis in advanced cases of pulmonary tuberculosis, where the discovery of the specific bacillus leaves no doubt. We will, however, consider the more initial cases which present but slight physical signs and few symptoms of the disease.

We must always bear in mind that in the great majority of cases pulmonary tuberculosis begins with one small focus that gives the patient no trouble, and which no physical examination, however minute, can detect. From this initial point proceeds, mostly very slowly, the diffusion of the disease in the way previously described; and the work of infiltration may have lasted several years before it has progressed sufficiently to disturb the patient and oblige him to call in the physician. It may thus happen that when the physician is first called in, the common symptoms and physical signs are already so manifest as to render the diagnosis plain, especially if the bacillus of tuberculosis can be discovered in the expectoration. But it may also happen that nearly

all the manifestations on which the physician founds his diagnosis are absent, for their development is very slow, as slow, in fact, as the march of the disease. In such cases one must be very cautious in saying that nothing is the matter, or that the individual is healthy.

Since the discovery of the bacillus there are those who will not admit the diagnosis of pulmonary tuberculosis, unless it has been discovered in the sputum. This is a fatal mistake. The bacillus may lie in the lungs many years without being expectorated, as has been already shown ; and when the bacillus is thus detected the pathological lesions are already considerably advanced and enlarged. And since it is of the highest practical importance to make the diagnosis as early as possible, it is important to emphasize the principal points that indicate an incipient pulmonary tuberculosis where the presence of the bacillus in the expectoration cannot yet be used as a test.

In the examination it is possible to detect a smaller expansion of the apex of one of the lungs, which can be better appreciated by standing behind the patient and looking over his shoulders to the root of the neck. This deficiency in expansion may also be felt by putting the hands in the sub-clavicular spaces and bidding the patient breathe slowly and fully. Remaining

behind the patient and putting the thumbs on the supra-clavicular spaces and the fingers in the infra-clavicular ones, one can well judge of the relative mobility of the two sides. A deficient expansion may indicate a beginning of tuberculosis at an apex even before a difference in sound is detected. Bidding the patient count one, two, three, one detects an increase of vocal fremitus where there is a tubercular infiltration or a caseous degeneration, but one must not forget that the fremitus at the right apex is normally stronger than at the left. Percussion generally furnishes us with very valuable data, but we must remember that sometimes injuries of some extent exist which are not very evident on percussion. To recognize the difference in sounds one must always compare the percussion of the two sides. One early symptom of great diagnostic value is a difference in resonance above and below the collar-bone. To further compare the two sides the patient is bidden to take a deep inspiration, and then hold his breath during percussion. By this method the difference is more perceptible. In thin individuals the percussion must also be carefully executed on the supra-spinal fossæ and in the interscapular spaces, as these are regions commonly attacked early by the disease. A weakening of the normal vesicular murmur forms one of the first local marks of tuberculosis upon auscultation ; but to be certain

careful comparison must be made with the corresponding point of the opposite side. In other cases one of the first symptoms will be a peculiar harsh breathing in marked contrast with the soft vesicular breathing of the neighbouring sound regions. In deep inspirations one also notices, not unfrequently a jerking or wavy rhythm, one or two irregularities in the same inspiration. As one of the first signs also, one may detect a slight pleuritic rub.

All these symptoms may be met with while there is no expectoration; the cough may be unimportant, limited to a few dry attacks, though it may be rather irritating, and without there being fever, perspiration, loss of flesh or other general disturbance of any importance. Now, when a physician detects a deficient expansion of one apex with increase of tactile vocal fremitus, with variation in sound on percussion, and a weakening of respiratory sound on auscultation, or the presence of a harsh breathing, the diagnosis of a tubercular area at an apex must be considered as of the utmost probability. If to these phenomena it be added that the patient has had a relative affected by pulmonary tuberculosis, or has nursed a consumptive patient, or has previously suffered from hæmoptysis, or even has been affected with pleurisy, the diagnosis of tuberculosis of the lungs must be considered as certain.

CHAPTER IX.

TREATMENT.

After having analysed the principal facts, anatomical and physiological, pathological and etiological, that characterize tuberculosis of the lungs, it will be easy to examine rapidly the various methods of treatment practised or advocated, and to come to a sensible conclusion on the benefits we may expect from each. It is not possible to allude to the innumerable special methods of treatment that have been adopted; space would not permit of it, and it would make a useless, nay, a hurtful digression in diverting the mind from the principal argument, for my desire is to draw attention to a method of treatment meeting as much as possible the requirements of our anatomical, pathological and etiological knowledge on which we have already dwelt, and one which may consequently give the maximum of good results that can be expected in the treatment of tuberculosis of the lungs.

REMEDIES GIVEN BY THE MOUTH.

Remedies given by the mouth are prescribed with two ends: (1,) To act curatively on the seat

of the disease and repair the damage caused by it ; (2,) To support the strength, to increase the appetite, and to control the extraordinary losses the patient has sustained, etc.

When I see a remedy of such a kind prescribed I receive about the same impression that I should if I saw the same medicine given by the mouth in order to cure a lupus on the face. What physician would expect to cure a lupus by any drug introduced into the stomach? It is the same, or almost the same, in pulmonary tuberculosis. Indeed, one may say that from the earliest days of physic, not one of the resources medical science possesses has ever availed to cure a single patient. Pulmonary consumption, like lupus, is simply a local malady produced first by a specific bacillus, and complicated later by septic micro-organisms. When these two causes are removed the malady ceases. Let us see then whether, notwithstanding the experience of many past years, we may hope to obtain some advantage by any addition to the number of remedies already employed. We have seen that the tubercle has no vessels, and not only so, but the pulmonary vessels that surround it are closed. If we inject a branch of the pulmonary artery we see that the injection, while it fills the capillary net that surrounds the tubercle, does not in the least penetrate the tubercle itself. We may therefore be sure that

whatever substance may be given by the mouth will reach the blood and with the blood be carried to the lungs, but it will never exert the least influence on the tubercle, because it cannot penetrate it.

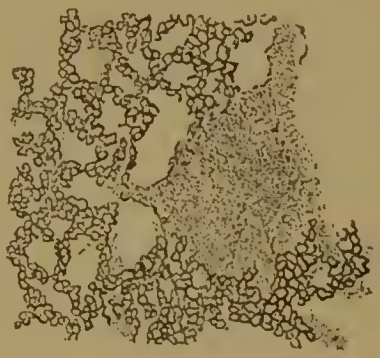


FIG. 10.

Gray miliary tubercle of the lung surrounded by a sound pulmonary tissue whose capillary vessels are injected. The figure shows that not the smallest part of the substance that entered the capillary vessels surrounding the tubercle would penetrate the substance of the tubercle itself.

Another point to be considered is that the bacillus of tuberculosis offers a considerable resistance to common disinfecting agents; it has been seen to survive for upwards of forty-eight hours in consumptive sputum that had been dried, then pounded and the powder treated with a solution of corrosive sublimate of 3 in 1,000; to resist during twenty-four hours the action of a solution of 1 in 20 carbolic acid, into which the pure culture had been plunged for a minute; the same thing happened with cresolina; cresol and

lisol have proved more active. These facts show that the bacillus of tuberculosis is endowed with considerable resisting power. How, then, can we hope that, introduced into the seven litres or thereabouts of blood that the body of an average man contains, so extremely attenuated a remedy can exercise any appreciable influence on this bacillus without first destroying the cells of the body itself which are more easily destroyed by the common disinfecting agents?

Again, let us remember a third point: When a volatile remedy is introduced into the circulation, it may in a limited degree, according to its volatility and the transuding power of the vapour pass from the capillary vessels into the pulmonary alveoli and be eliminated with expired air. But this elimination at the best is very limited. For instance, creasote, which is one of the remedies oftenest employed, enters the alveoli very little, if at all; it continues rather to circulate with the blood and is finally eliminated with the urine. We have proof of this in the fact that when we have administered large doses of creasote, or if we continue it long, the urine assumes a brown colour as if smoked—an important sign that warns the physician to stop the drug.

To conclude, we may affirm that the medicines swallowed are diluted in the great blood-mass of the body, and reaching the lungs thus are unable

to penetrate to the tubercle because it is itself without vessels, and the surrounding vessels are closed; moreover, could these medicines even touch the tubercle the effect would be small since they would still be carried onward in the blood stream.

However, we are not to believe that they are wholly ineffectual. It is a fact that creasote and guaiacol influence the fever and perspiration, increase the appetite, and cause a general amelioration of the conditions. Probably the reason is to be found in the way in which creasote acts on the toxins formed in the tubercular lesions, which are absorbed by the blood. Thus it would act, not on the specific micro-organisms themselves, but on their products. This benefit might continue so long as the tubercular lesions were not sufficiently numerous to furnish such a quantity of toxin that the largest dose of creasote that could be administered with safety would not overcome its action.

Again, not only creasote, but all balsamic substances, stimulating the nervous terminations in the bronchial mucosa, increase the circulation that the action of the disease has made sluggish around the tubercular foci. In consequence the functionality of the local glands is renewed, and the bronchial secretion which has been tenacious and sticky becomes looser and is more easily expelled.

Arsenic which was widely employed some years ago acts partly in this way, but perhaps its more important effects are produced on the bronchial epithelium, quickening the elimination of the epithelial cells and lessening the risk of bacilli finding a nidus in them. This is important, because by preventing the retention of the bacilli on the epithelium it checks the formation of new foci.

Other remedies reduce the sensitiveness of the bronchial nerve-endings. Thus the various irritations and injuries produced in the respiratory regions during the course of the disease are not so acutely felt, and hence the cough is appreciably diminished. Opium and its compounds may be placed at the head of such remedies, and in some cases they are highly serviceable. Without insisting on minor and well-known facts, I think we may conclude that from the class of remedies administered by the mouth we have little more to hope for in the future than has already been attained.

The slight advantages obtained by these drugs are often counterbalanced by the bad effects they may produce in other directions. The prolonged use of some of them, for instance guaiacol, or creasote which contains 60 per cent. of guaiacol, often disturbs digestion. Not only does the appetite of the patient fail, but sometimes

he experiences a positive loathing for food, which he neither digests nor assimilates, so that the want of food forms one more element to increase his loss of flesh and vitality. Nutrition failing, we lack a powerful aid to recovery, so powerful indeed that probably the mischief effected by the remedy is greater than the advantage it brings.

In conclusion we affirm that the anatomical conditions, the pathological changes and the root cause of the disease clearly prove that, while no curative action can be expected from a remedy either swallowed or introduced, for instance, hypodermically, yet the harm derived from remedies may be sometimes far from trifling. It is therefore desirable that medical science should cease looking for curative remedies of this nature. Such remedies should be confined to obtaining only those advantages and ameliorations of the diseased conditions that may be expected from them, such as soothing the cough, increasing the appetite, maintaining the strength, producing sleep, etc.

ANTI-TUBERCULAR SERUMS.

In a preceding chapter I have called attention to the advantages that might be derived from a serum, if ever discovered, that should possess anti-tubercular properties. I say *if ever*, for keeping to what Landouzy said at the last Congress

on tuberculosis in Paris, we are compelled to acknowledge that, neither in Italy nor out of it, does there exist to-day any such serum.

Leaving other so-called anti-tubercular serums that have been recommended for the cure of tuberculosis, I shall say a few words on the serum of Maragliano, which is the only one we employ. Before doing so, however, I wish to mention an important fact that should never be forgotten; and that is that it is not the extent nor the number of the lesions of the lung which kill the consumptive patient, but rather, and one might say solely, the nature of the injuries. The natural course of tuberculosis is so slow that if the atmospheric pyogenic germs did not come in to render the lesions septic, thus to hasten the work of destruction, and cause death by the more or less slow and repeated toxæmias, pulmonary tuberculosis would probably not be a very fatal disease. We see in fact how slowly lupus progresses, since its lesions are not all exposed to septic infection, as they are in pulmonary disease.

And if we remember besides this, that when we discover a pulmonary tuberculosis, the work of infiltration is usually already far advanced, we come to the conclusion that any anti-bacillar serum will always have but a very limited action. Such a serum might be of use in the very first stage of the malady, but not in the second, nor in the third.

To know whether a serum is really anti-bacillar, in man, would seem to require a very simple test: examine its action on a lupus. If the discoverer of an anti-bacillar serum could publish statistics showing a series of recoveries from lupus through its means, one might conclude that the slight improvements we have mentioned above are to be expected.

This result, as far as I know up to the present, neither the serum of Maragliano nor of anyone else has given.

The serum of Professor Maragliano has been conscientiously tried in the clinic of Professor Murri at Bologna, and the result of the observations was published at the beginning of this year by two medical assistants, Drs. Dagnini and Silvagni. The observations were made on twenty-five patients, and the conclusions come to were that in the feverish forms of lung tuberculosis, the serum of Maragliano is of no avail; and in the apyrexial forms it does not add any advantage to those which are given by rest in bed, by the relatively wholesome conditions of the hospital, and by good nutrition.

OPEN-AIR SANATORIA.

In the little village of Sutton Coldfield (Warwickshire, England), about the year 1830, lived George Bodington, a medical man. He was one of

those close observers who could analyse facts, when he found them, and act upon his knowledge fearlessly without allowing current opinions to over-rule his judgment. In 1840 he wrote a pamphlet entitled: "An Essay on the Cure of Pulmonary Consumption, on Principles Natural, Rational, and Successful," in which he stated truths that might now be quoted as belonging to the present question. Speaking of the importance of pure air he says: "To live in the open air, to constantly breathe pure air without dreading either wind or bad weather constitutes an important and essential remedy to stay the progress of pulmonary consumption; a remedy against which a groundless fear prevails, that the consumptive patient may catch a cold. Farm men, shepherds, peasants, etc., are rarely affected by consumption because they constantly live in the open air; while the inhabitants of cities and those who live long in shut-up rooms, or whose calling obliges them to spend many hours of the day in closed spaces are often victims of it. In the treatment of this malady the habits of the latter must be so changed as to make them resemble, as much as possible, those of the first category of individuals so far as regards their living in the open air, if we wish them to recover. . . . I will now speak of the most important remedy for the recovery from consumption, that is to say, the free use of a pure

atmosphere. The sick person should inhabit a well-aired house in the country. . . . Fear of the weather should never prevent him from living in the open air ; if the weather is wet and rainy he must use a covered carriage with open windows. The cold is never too intense for a consumptive patient. . . . The apartments should be kept cool and well-aired, so as to resemble the free air outside, pure air being employed in the treatment as much as possible."

He had observed the mischief which results from shutting up patients for fear of admitting air, as may be inferred from what follows: "A manner of treating the disease is that of shutting up the patients in a room and forbidding the entrance of the free air from outside, thus obliging them to inspire and expire over and over again the same foul air, corrupted by the emanations of the sick person. . . . This is indeed a method of treatment founded on the most mistaken principles."

Speaking of nutrition he says : "To a patient who daily loses weight under the influence of a *wasting disease*, I must recommend a diet composed of meat and farinaceous substances with some wine, according to the general conditions of the patient. As will be seen hereafter, I have prescribed a nourishing diet moderately stimulating, with great success ; without it I do not think the

other means would have proved so efficacious, or the treatment complete. . . ."

In order to understand the effect produced by this essay, we must go back to 1840, when physicians dreaded air as their worst enemy, when the first care of the doctor was to warn the patient against every gust of air; when the unfortunate consumptive patient was kept months and months in a room with a stern prohibition against opening the windows; nay, woollen hangings were nailed against doors to ward off the little air that might enter when the door was opened; when every illness was treated with a severe diet of beef-broth, no wine, no meat, etc.; so that it seemed as if Medicine was striving to hasten the progress of the illness, contributing to it with frequent bleedings, antimonial preparations, and free use of calomel.

This book met with the most bitter and fierce opposition, so much so that its author was considered as little better than a lunatic, and undeserving of serious attention. However resolutely he maintained his position, however firmly he supported his ideas, disapproval so universal drove the patients from his establishment, where, several years previous to the publication of his essay, he had acted according to his principles and effected many cures. He finished by giving up the curing of consumption, and transformed his hospital into a lunatic asylum.

But all did not take things so lightly. A great physician of those times, John Hughes Bennet, approved of the teaching of Bodington, and in 1857 Dr. Benjamin Ward Richardson, in the January number of the *Journal of Public Health*, gave great publicity to the theories of the poor country physician, thus arousing the attention of the medical world. Two years later we see in fact, Dr. Brehmer, of Görberdorf, starting the first sanatorium where, with organization, the principles of Dr. Bodington were acted upon.

From that day the idea of sanatoria has steadily gained ground. They are now multiplied in all parts of the world. In 1896 the Berlin Brandenburg Society for the construction of sanatoria was formed; at the same time the sanatorium of Grabowsee was built, and finally a central committee was constituted for the erection of sanatoria, whose president was the late Prince of Hohenlohe, chancellor of the Empire.

There exist now in Germany thirty-three popular sanatoria, and at the last International Congress on Tuberculosis, held last May at Berlin, the formation of a committee was announced to erect a monument to Dr. Brehmer, "the leader of the modern movement in favour of sanatoria, and the founder of the open air cure for tuberculosis," while the poor country doctor of Sutton Coldfield who, at an earlier epoch, had the courage

to oppose himself to a mass of fatal errors, and to sow the first seeds of a rational treatment, for which he reaped scorn and contempt, with grievous material losses, now lies wholly forgotten; few even are aware that he ever lived, and that the modern stir about the "open air cure" was originated by him.

The treatment in sanatoria, that is to say, by life in the open air, has proved by facts that pulmonary consumption is curable. The unfortunate consumptive patients formerly all died, because they were constantly placed in such conditions that recovery was not only impossible, but the progress of the disease was actually hastened, rather than checked.

A consumptive patient, before being admitted to a sanatorium, must undergo an examination, with a view to see that the malady is not sufficiently advanced to prevent hope of recovery; before being finally accepted, doubtful patients must be kept under observation for a time with a view to seeing what influence the new treatment has on the course of the malady. The principles acted upon are the same that Bodington tried, and they consist in constantly breathing a pure air day and night, and feeding on a nourishing diet.

In Colorado, in the United States, which may be considered as the Riviera of America, many sanatoria are simply composed of tents, in which the

patients spend the night and those hours of the day when bad weather does not permit them to stay in the open air, the results being excellent. The use of the sanatorium is more efficient the more the purity of the air in the rooms approximates to that of the open air. Therefore every effort is made to obtain a perfect ventilation by day as well as by night. The cold, which in some Alpine regions in winter may reach to 4 or more below zero, is counteracted by warm wraps and good fires in chimneys: the latter help to produce good ventilation, but one should never avoid the cold by excluding fresh air. Feverish patients are kept in the open air under a verandah, reclining on special chairs, covered with rugs; those who have no fever may take walks in accordance with their condition. The stay in the open air is certainly very favourable.

All this is highly rational, and as soon as it was tried, gave the same good results that Bodington had already obtained in his little sanatorium. Sick people who come to the sanatorium depressed and weak, very often, after a fortnight or a month acquire again their normal temperature, their appetite revives, digestion is improved, they have quiet sleep, night sweats cease, and they increase in weight and look healthy. Unfortunately there are others who seem to derive little good even from the sanatorium, but these are not numerous. How

is this great improvement in the consumptive brought about? How does fresh air act on the lesions?

The answer is not difficult for anyone who knows their pathology and condition, when the patient arrives in the sanatorium. Sometimes the lesions are not large, only an infiltration of an apex with very small tubercular cavities scattered on the infiltrated portion. Each of these small cavities, of the size of a millet seed or little more, forms a centre of septic inflammation, harbouring quite a nest of pyogenic streptococci, and causing a considerable degree of infection with high fever, night sweats, etc. These are the cases which generally feel at once the good effects of the sanatorium. After a few days of breathing pure air, the pyogenic infection of the small lesions not being fed by new impurities, subsides, and with this the predominating phenomena, while the general health improves. All this is natural, for the infiltrations and the minute cavities do not of themselves cause any deterioration of general health, the disturbance only beginning when these spaces grow septic, as they must necessarily do in the great centres of population, where the air is excessively charged with micro-organisms, and especially when patients are enclosed in rooms, where the atmospheric impurity is intensified very considerably. In a room only ventilated by day,

with the windows shut all night, and in addition freely exposed to foul smells from an ill-kept closet, a dirty sink, and a hundred other sources of impurity due to putrefaction of organic substances, we shall have the most favourable conditions for the introduction into the little pulmonary tubercular lesions of an enormous quantity of septic micro-organisms, capable of producing high fevers, even from the smallest lesions, and of causing a rapid destruction of the pulmonary tissue. A prompt change must be made to hinder the introduction of septic micro-organisms, the sole cause of these mischiefs.

In such cases the effect of sanatoria is marvellous. But as soon as the patient returns to town and resumes his customary life, the fever re-appears and he reverts to his former condition. This is what is continually seen, and is to be expected, for in the sanatoria the lesions are not healed; they merely become aseptic, ceasing to cause a disturbance to the general health, and possibly healing spontaneously with time, if new septic infection from without does not awaken the dormant foci.

A great advantage of the sanatoria is also the education that the patients receive. There they are convinced that perfect ventilation and pure air are their principal remedies; they never expectorate but in their own spittoon; they learn that the place they live in must be scrupulously

clean, since every organic substance existing in it may become a source of putrefaction and corrupt the air. When they return to their homes they continue these habits to their great advantage. I have never met a patient who, after spending some time in a sanatorium, did not continue to sleep with open windows. Continuing this good habit, it sometimes happens that the pulmonary lesions are not re-infected, the patients living under good general conditions for many years without a sign of illness. It may also happen that a spontaneous recovery occurs in the manner indicated in the preceding chapter, especially when the lesions are not large. This is what a sanatorium can show, and what the so-called "open air treatment," which in some localities is gaining ground, can do; in fact, the latter may in great part be a substitute for sanatoria.

The open air treatment may be carried out anywhere by applying the rules of the sanatoria. It is for the most part easily applicable to hospitals, and often to private houses. Hospitals have generally a garden. The method followed would be to take the patients with fever into the garden, as far as possible from the building, and keep them all day in bed under a portico to shelter them from bad weather. In the evening they would return to their room, which should be well aired, and kept night and day with open windows.

In private houses it is not difficult, in most cases, to apply such a method of treatment, especially if the family of the sick person possess a country house where the patient can spend the whole day out of doors, either in bed, sitting, or taking strolls; but during the cold season, almost always in bed.

We learn the good results obtained by this method of treatment from a recent monograph by Dr. Bardswell, published in the *Lancet* of November 7th, 1899. In the month of May the Royal Infirmary of Sheffield adopted the open air treatment for all its consumptive patients, without choice or distinction. From that date to October, out of thirty cases treated, six may be considered recovered, sixteen greatly improved, seven somewhat better, and one only did not derive any benefit.

Here is the condition of some of the patients:—

No. 1. Age 16. When about to be admitted he was almost dying; much diarrhœa, high fever, large lesions in the lungs, with expectoration full of bacilli, no appetite, laborious digestion.. In the open air all the phenomena improved at once, so that after 38 weeks his condition was as follows: Marks of extensive fibrous cicatrization, no fever, good appetite, bacilli reduced in number; he can walk, even as far as five or six kilometres a day, his weight increased by 7½ kilogr.

No. 2.—Age 24. Active and widespread injuries in the two lungs with cavities; numerous bacilli and albuminuria. After 29 weeks' treatment the pulmonary conditions are much improved, the cavities are dry and healing, the appetite good. The albuminuria remained, and the patient could resume his work. Increase of weight 6½ kilogr.

No. 3—Age 37. Hæmoptysis, active infiltration in both lungs, no appetite, and laborious digestion; presence of bacilli. After 17 weeks of treatment the lungs are much drier and likely to recover, the appetite is good, the digestion also; no sweating. Weight increased by 9½ kilogr.

No. 11.—Age 17. Cavity in the right apex, active lesion in the left lung, slight fever, good appetite. After about 4½ weeks, very rapid improvement in all conditions. Increase of weight 5·70 kilogr.

No. 12.—Age 34. Recent hæmoptysis; large infiltration in the right lung, fever, want of appetite. After treatment for 4½ weeks, no fever, good appetite, general improvement.

The statement furnishes an abridged account of 12 patients, arranged according to the length of the time in which they followed the treatment. For brevity's sake I have only related the story of the three first and two last, for they all resemble each other.

If such results were obtained at Sheffield, which

is a very crowded city (204,677 inhabitants), full of commerce and industry, lying low, etc., why should we not derive the same benefits in our hospitals, which are generally under better conditions as regards climate, and position, and free from the crowding, and foulness of the atmosphere from the presence of factories of all kinds; why too could not such a method of treatment be adopted even in families, as far as practicable?

Before passing on to other points I wish to allude to the unbounded enthusiasm of the day for the erection of sanatoria. What has been already stated will make it clear that no one can well be more in favour of their erection than myself; whether it is an unmixed good that the whole of the public anxiety for the assistance of the consumptive poor should be given to sanatoria, as if such adjuncts were the only means of curing, is, however, doubtful.

The erection of a sanatorium is very expensive, but its maintenance is more expensive still. If we calculate the consumptives of Italy at 250,000 we are below the number. Of these more than 100,000 are poor and should be transferred to sanatoria. We should therefore require to build such a number of sanatoria as could hold and keep for a whole year 100,000 patients. If we reckon every patient in a sanatorium to cost 3 francs a day, we are very moderate; therefore the maintenance, without

reckoning the expenses of erection, would cost over 100,000,000 francs a year.

The total number of the sick that are sheltered daily in the hospitals of our kingdom is about 30,000, and we should require to build sanatoria in sufficient numbers to be able to receive three times as many patients as there are for all kinds of illnesses in all our hospitals. Such a scheme is so immense that it becomes impracticable, and the question arises whether one cannot devise something more useful, and capable of immediate application. The least that should be done in our country as regards sanatoria would be the erection of one in every province, but this would be of little use, for the number of needy consumptives in every province is always infinitely greater than could be received into any sanatorium. For instance, in the province of Perugia, which is by no means one of the worst affected, there die on an average from this disease 500 patients a year ; but then 1,400 more die of bronchitis, of whom at least 1,000 may be considered consumptive. That is to say, we have annually 1,500 deaths from consumption, which would correspond to about 4,500 living consumptives.

It is manifest that the cost of sanatoria capable of giving anything like this accommodation would be absolutely prohibitive. My own opinion is that, without giving up the idea of sanatoria, for which

there will probably be a constant and steady demand, it would be better to begin by inaugurating "the open-air cure" at once in hospitals, and at the same time to endeavour to educate our people in the doctrine that the greatest peril from tuberculosis lies in badly aired apartments. I quote on this head the following figures: The mortality from tuberculosis in England in the decade from 1851 to 1860 was 26·79 for every 100,000 persons. From then to now the mortality has steadily diminished, so that it was 24·75 in the next decade; 21·16 from 1871–80; 17·32 from 1881–90, and only 14·93 in the five years from 1891–95.

As regards the great struggle with disease that is being fought in our days in all civilized countries, and especially in England, tuberculosis was only recently dealt with; at first, other infectious maladies were combated (small-pox, typhus, diphtheria, etc), but the knowledge of tuberculosis was so slight that it was impossible to find a rational means of preventing it. How, then, has England obtained the enormous diminution of nearly one half in the death rate from this disease without any effort specially directed against it. The fact is very easily accounted for. In no country in the world have the true principles of wholesome hygiene been so thoroughly understood by the population as in England, therefore no other country has made such progress in the healthy construction

of dwellings. That a wholesome dwelling is the first condition to health in a family—the most powerful means of escaping disease—is now generally acknowledged in England by every social class, so that when the head of a family seeks a change of residence, the first points for consideration are usually the sanitary conditions of the new dwelling, without which he knows that disease cannot be avoided. Unhealthy houses of necessity disappeared before this pervading feeling; and, in proportion, tuberculosis and other maladies diminished apace.

If practising physicians and, above all, village doctors, would wage a hygienic war in Italy, if they would make people understand the importance of fresh air, the perils of all kinds, and above all of tuberculosis, that threaten those who breathe foul air, the air of privies and of dunghills, the air that has not been renewed in bed chambers, and so forth; if they could persuade our people at least to sleep with open windows, as well in winter as in summer, there would be immediately such a diminution of tuberculosis, not to speak of other diseases, as could not be obtained by the erection of many sanatoria.

TREATMENT BY INHALATION.

We may say that Medicine in all ages has endeavoured to fill the lungs with volatile

substances with the intention of healing their lesions, and therefore the history of inhalations is as old as Medicine itself. Homer descanted on the healing power of sulphur, Galen sent his consumptive patients to Mount Etna and Vesuvius to inhale the air impregnated with sulphur. The inhaling of the fumes of sulphur always claimed the attention of physicians, till the knowledge acquired in the last fifteen years drew it to other and surer agents. The famous English doctor, Edward Jenner, the originator of vaccination, recommended the inhalation of the "vapour of tar, heated and diluted with essence of turpentine," in the hope of killing the parasite to which he attributed the malady, and which he believed to be very similar, if not identical, to that of the hydatid cyst. He compared the tubercle to the oak-gall, or to the hairy excrescence that is to be observed on the branches of the dog rose, which, in both cases are due to the "irritation produced by an insect." A great number of substances had been already used for inhalations in the last century, and Laennec, in his classical treatise on "Auscultation Médiate et des Maladies des Poumons et du Cœur" observes (vol. ii., pages 182, 183,) how the experiment has been tried of creating for the patient an artificial atmosphere of divers gases or vapours, derived from various substances, such as aromatic plants and balsamic myrrh, benzoin, etc., or petroleum,

tar, ammonia, resin, sulphur; and how an attempt has also been made to "obtain the inhalation of divers gases, such as oxygen, sulphuretted hydrogen, and carbonic acid, by means of a convenient apparatus." In the same volume much is said about the inhalation of chlorine. From this time till the discovery of Koch, inhalations of different kinds have been recommended by all authors, and several volumes have been written on the treatment of pulmonary diseases through inhalations. All these recommendations were made without any scientific basis, but solely on the evidence of the good results that were occasionally observed to follow the one or the other inhalation.

After the discovery of Koch, the hope of killing the bacillus of tuberculosis caused the number of apparatus for inhalation to be multiplied, as well as the number of the substances for inhalation, so that, at the present day, no writer on pulmonary diseases fails to mention inhalations, either blaming or praising them, according to the result of his own observations; and we may say that to-day there scarcely exists any antiseptic volatile substance that has not been tried in pulmonary inhalations. Many methods, too, have been devised for this purpose, some good enough, while others proved of little service.

Among the latter, let us place all inhalers that

are exclusively applied to the mouth. The nostrils are the true respiratory passages, and an effective inhalation will never be obtained by the mouth only; worse still, if the instrument is to be held in the mouth itself, or to be used like a pipe. In such a case, when breathing, the patient must make a slight effort to swallow the vapours, and in consequence the inhalations must be very imperfect and broken.

Other useless inhalers are those after the model of the well-known apparatus of Siegel, with which by means of an alcohol lamp a spray of liquid is produced for the patient to inhale. It is found that substances thus applied do not reach even the bottom of the wind-pipe. Therefore, they are not only of no use, but very harmful, for they delude both doctor and patient into the belief that they are combating the disease, and they do not look for other remedies, losing thereby precious time, during which the malady has leisure to progress. Another method of no use at all, if not really harmful, is that of developing antiseptic vapours in a small room, where the patient is compelled to spend the greater part of the day in breathing the vapours. Setting apart the fact that such a method is very annoying to the patient, it offers great obstacles to his recovery, as for the best part of the day he is compelled to breathe foul air. One must never

cherish the wild hypothesis that a little tar or other antiseptic vapour can ever purify the air of a narrow room, where individuals *affected by a pulmonary malady* stay many hours with closed windows.

The idea that has prevailed since Koch's discovery in advising inhalation of microbicidal substances, is to kill the bacilli of tuberculosis in the lungs, and authors, with rare exception, limited themselves to prescribing inhalations for a few hours a day, deeming this enough. The principle of inhalation to be successful must be quite different from this, and deserves to be treated at some length.

PRINCIPLES TO BE ADOPTED IN INHALATION.

Let us state once more the most important fact that must guide both patient and physician in the treatment of pulmonary consumption—a fact about which there is not the slightest doubt—that is, the patient does not die of pure tuberculosis, but always in consequence of the infection of the lung lesions by atmospheric micro-organisms. Perhaps no individual ever died of pure pulmonary consumption.

Almost the same affirmation could be made of surgery prior to the introduction of antiseptics. In those days, of which the young practitioner can now form but little idea, the slightest operations

were often followed by death. In the surgical clinic of Professor Titus Vanzetti, of Padua, where I completed my studies, I saw patients die in consequence of a simple amputation of an arm, of a foot, or for the excision of a breast, and once I saw a strong, hale man of about forty succumb to a simple incision made in removing a portion of the infra-orbital nerve, an operation which was of little more importance than a bleeding. Yet Professor Vanzetti was justly considered one of the best surgeons of his time, in Italy as well as abroad, and his wards were kept with as much cleanliness as any other. Death did not result from the slight injury of the operation, but in consequence of the infection of the wound by the common septic streptococci of the air which produced blood-poisoning, with rigors, fever, abundant sweating and death. With the introduction of antiseptic surgery this cause of death disappeared, and our surgeons not only saw nearly all their patients recover, 90 per cent. of whom formerly would have died, but they can now perform formidable operations which were then beyond even contemplation with the happiest results.

Death from pulmonary consumption results from the same secondary cause that it did after these old style surgical wounds; it is neither the extent nor the importance of the injuries that kill, but the intensity of the toxæmia originated

in such injuries. Therefore, in the treatment of tuberculosis we should be guided by the same principles which govern antiseptic surgery. These principles lead us not only to remove the cause of death (septic infection), but to strive to destroy the specific bacilli of tuberculosis and to completely cure the disease.

We thus conclude in the clearest manner that the only really rational principle by which to treat tuberculosis and on which we may fully rely, when the local conditions are not yet so far advanced as to render recovery absolutely impossible, is the same which guides the surgeon in the antiseptic treatment of wounds. We must follow as much as possible the exact rules the surgeon observes and always bear in mind the precepts that have been laid down for him during the last five and twenty years, and must apply them, as far as the anatomical and pathological conditions allow, to the treatment of pulmonary consumption.

Is it possible to do this in such a way that we may reasonably hope for success? The first natural suggestion is the idea of introducing into the lungs by inspiration volatile antiseptic substances, that may disinfect the smallest infundibular and alveolar cavities as one would disinfect a room after an infectious disease. However, a difficulty which at first sight seemed insuperable was urged against this idea; in *lupus*, it was said, you have a

cutaneous lesion of the same nature as that contained in the lung, a wound that is under your direct control but over which no known antiseptic will give you any great advantage. The *lupus* does not stop notwithstanding the antiseptic, applied directly and in a concentrated form; what hope can you have then, for the lung where the injuries are only accessible to gaseous substances diluted with air?

Let us refer again to the anatomical structure of the lung, as described in earlier pages. We have seen that it is formed of a number of very minute lungs or pulmonary lobules (of the size of from 2 to 4 mm.), each of which has a tiny bronchus that divides into the alveolar canals and the infundibula, on whose sides are the alveoli or air cells.

The septa which separate the canals, the infundibula and the pulmonary lobules are extremely thin, as is evident when we remember that in this very tiny organ, the lung lobule, several alveolar canals, many infundibula and numberless alveoli are to be found. Therefore we may rationally hope that by continuing to introduce into the lungs a volatile antiseptic substance almost twenty-four hours daily during many months at a stretch, and sometimes during years, at the rate of about 25,000 inspirations per day, we may succeed in disinfecting these very thin septa of elastic and

connective tissue which unite to each other the various pulmonary structures (pulmonary alveoli, infundibula, alveolar canals, pulmonary lobules and bronchi).

This does not take place in *lupus* because the bacilli are deeply embedded in the skin and even in the underlying tissues. And whatever concentrated antiseptic remedies we may apply to the external lesion they cannot penetrate a millimetre into those tissues. We may obtain a slight improvement through the action they produce on the bacilli at the surface, but the deeply-seated ones must remain unaffected, and the disease continues. On the other hand, when we employ the cautery and penetrate widely and deeply into the diseased tissues, the bacilli are killed, and recovery follows.

This objection, therefore, simply shows that the anatomical structure of the pulmonary organs lends itself suitably to antiseptic treatment, while the region where the lupus settles does not.

Having thus established the possibility of antiseptic treatment, we must keep in mind that to be efficient it must resemble as much as possible the usual treatment of wounds. It is of the first consequence that it should be continuous: no surgeon would dream of leaving a wound open to the influence of septic atmospheric agents during many hours of the day. Inhalations, therefore, if they are to be of service, must

continue day and night, only ceasing at meal times and when absolutely required by the necessities of life.

These continuous inhalations form the basis of the treatment: they will therefore be considered in a separate chapter.

CONTINUOUS INHALATIONS.

The method I adopt is this: I use a pyramidal mask having a base shaped like a horseshoe and

Fig. 11.

constructed of wire netting with a mesh containing ten divisions to every square centimetre. The posterior portion of the sides is open and is applied to the mouth and nostrils so that the apex of the pyramid rests against the root

of the nose, the sides and base of the pyramid forming thus a cage, closed on the under side and base by resting on the face of the patient. This mask is covered a good way down the inside by a thin layer of sponge, in such a way as not to touch the nose. The open edges which lie against the face are bound with glove-kid and are furnished on each side with strings which serve to fasten the inhaler against the face. Instead of the strings rubber straps may be used fitting behind the ear.

From 30 to 40 drops of an antiseptic liquid are poured on the sponge, and the inhaler is then placed in position in the manner indicated. Every two hours the 40 drops are renewed, and during the night whenever the patient awakes.

The antiseptics I have used are: Volatile oil of turpentine, carbolic acid, creasote, camphor, thymol, thymol camphor, menthol, volatile oil of mint, and eucalyptus oil; but for many years now I have preferred a combination of creasote, alcohol and chloroform.

These three liquids form a volatile compound of not disagreeable odour in which chloroform is perceived first, but the smell of creasote remains longer. Patients easily accustom themselves to it, so that after a few days they do not feel any loathing, but generally rather like it.

Creasote is employed for its highly antiseptic

properties. Chloroform is also used for the same reason and because, being very volatile, it readily carries the creasote held in solution to the ultimate bronchial ramifications and to the alveoli; from its well-known effects on nerve, it is not improbable that it also affects the terminations of the bronchial nerves, blunting their sensitiveness and thereby diminishing the tendency to cough. Alcohol acts principally as a diluent, the mixture of pure chloroform and creasote being too strong; alcohol, too, is volatile to some extent and endowed with strong antiseptic properties. This mixture seems fairly to meet the indications of the malady.

In the communication I made last September to the Umbrian Medical Congress, I said I had employed for six months a mixture of antiseptic liquids, which perhaps was quicker in effect. Concerning this I hope to speak next year when I have gathered more material on which to found a positive opinion.

Two important questions arise here: (1,) Does this antiseptic mixture really find its way to the smallest bronchi, to the infundibula and alveoli, so that it may come into contact with the injured tissues? (2,) If it really reaches them, is there no danger of its being absorbed by the blood that circulates in the thick capillary net of these structures, and being thus brought directly

into the arterial circulation, may it not interfere with the functions of the more remote vital organs which this blood should nourish?

The first question may be answered unreservedly in the affirmative. We have proof that chloroform penetrates into the alveoli by its great diffusive power in the fact that as soon as a little quantity of it is inhaled from a pocket-handkerchief the effect is felt at once in the brain, in cerebral dizziness, showing that the chloroform penetrates really into the alveoli, where it is absorbed and brought into the circulation. One cannot imagine that the chloroform that holds the creasote in solution parts from it and penetrates alone into the farthest pulmonary recesses. Therefore we may be sure that the vapours of the mixture are carried into the pulmonary lobules, so that the whole lung, as the patient continuously inhales these vapours during nearly twenty-four hours of the day, comes to hold in its alveoli a considerable quantity of the vapours diluted with the oxygen and nitrogen of the air. In other words, the pulmonary tissue is constantly permeated with an atmosphere of creasote, alcohol and chloroform mingled with atmospheric air.

To the second question the experience of the last sixteen years furnishes an answer. Chloroform is certainly absorbed and brought into the circulation as already stated; but its quantity is so

slight and it is so quickly eliminated that no mischief is to be feared. The creasote, which is of a much less volatile nature than chloroform, when it reaches the infundibula is unable to penetrate the capillaries and to mix with the blood. The same thing takes place with the creasote as with the nitrogen of the air; the latter penetrates the infundibula and alveoli, but it is not absorbed by the blood, as has been shown in previous pages.

If creasote were brought into the circulation, we should observe its effects on the kidneys and urine.

We have already stated that creasote given by the mouth may, after a short time, give a smoky appearance to the urine. I have only observed anything approaching this condition, as a result of inhalation, in one of my patients, a woman who had used the inhaler seven years. This patient's urine first became a dark brown, and this was followed by a real hæmaturia of short duration. But I cannot think that it should be attributed to creasote after its use for seven years. I have had many patients who have used the inhaler for three and four years without any traces of albuminuria or smoky urine. Therefore I conclude that creasote, penetrating in the form of vapour to the pulmonary lobules, remains there without entering the circulation, just as does the nitrogen of the air.

We have seen that creasote given as a liquid is absorbed by the blood, and when by this channel it is carried to the lungs very little of it remains in the pulmonary alveoli, for it continues to circulate in the blood, to be finally eliminated by the kidneys. On the other hand, the creasote introduced into the alveoli by inhalation is not absorbed by the blood, but remains in the air of the alveoli themselves. Creasote, then, taken into the stomach does not stay in the injured regions, but is carried to distant and sound organs where it produces serious mischief when the doses are high or too prolonged; but when inhaled it remains largely where it is wanted without entering the circulation; we, therefore, need not fear mischief to other sound organs.

We have already pointed out that tubercles are non-vascular, that the neighbouring blood-vessels are hindered from interchange with them, and that when their contents have been softened and expelled through the communicating bronchi there remain little cavities enclosed by the surrounding blood-vessels, while the bronchi, by which their contents have been discharged, communicate freely with them. Thus, while drugs circulating in the blood-vessels cannot have any effect on the tubercular lesions, remedies introduced by inhalation not only permeate the tubercle, but also fill the minute tubercular cavities and act as

bactericides on the micro-organisms that are present. Further, the septic micro-organisms of the air probably do not penetrate *at once* into the tubercular area, but are retained awhile by the action of the ciliated epithelium, by the mucus with which the tubes are lined, and by the fact that the minor bronchi only open in the act of breathing, and therefore these micro-organisms may be killed by the action of the antiseptic before they reach the injured tissues, and may also be gradually destroyed in the lesions themselves by the diffusion of antiseptic vapour. We must, however, remark that when we are dealing with large cavities filled with a liquid abounding in organic detritus, thick and purulent, a liquid highly favourable to the proliferation of such septic germs, the action of the antiseptic is very slow, and more so if the cavities are numerous.

Nay, all who have had experience of these lesions and opportunities of examining tubercular lungs after death will know that sometimes the lung presents in some parts such a number of cavities that a portion of it has no communication whatever with the external air. To this no remedy can reach, not even by inhalation, so that the work of destruction goes on unchecked. This may happen, too, though the extent of injured lung is not large, which explains how sometimes, notwithstanding the uninterrupted inhalations, the fever continues

even when the injury is limited; while in other cases, though the lesions spread over a whole lung, there may be a wonderful improvement at once.

RESULTS OF THE TREATMENT.

My use of uninterrupted inhalations dates from 1883, after the discovery of the bacillus of tuberculosis by Koch in 1882. The population of Serina and Oltre il Colle (Bergamo), of which I was then the district physician, remembers those cases, which were considered extraordinary because the patients were obliged to wear "a muzzle." The results were very encouraging. I constantly observed extraordinary ameliorations of the symptoms promising a prompt recovery. But as soon as the patients lost their cough they forsook the treatment and after a few months had a relapse.

These were fatal, not from the hopelessness of the cases, but because the patients allowed themselves to be convinced by their friends that their cure was impossible, and further that the medicines "burnt inside." One knows the caustic nature of the carbolic acid I employed at that time, and the patients frequently felt a slight effect from it on some parts of the face, especially the tip of the nose. The red marks gave rise to the notion that the medicine "burnt inside."

In January, 1885, I came to teach medicine in

this university, and in the following February I was called to visit, in consultation with Dr. Revoira, a patient who had had violent hæmoptysis four days previously, which was repeated three times. He was a young man of 30, of fairly strong physique, and presented a wide-spread infiltration in the right lung, from the apex to the base, and also at the apex in the left one. He had very high fever, abundant perspiration, emaciation, lack of appetite, and difficulty in digestion.

When we were going out, the sister of the patient having asked whether there was any hope, the attending physician answered that not only was there none, but that only a few days of life remained, and it would have been better had he died during the attack of bleeding, as it would have terminated his sufferings. I interfered, and said that, while I joined with the doctor in believing the malady to be very serious, I could not say it was hopeless. I examined the sputum and found the bacilli of tuberculosis.

The treatment was at once begun, and the result was that in the following month, September, on the occasion of the Medical Congress at Perugia, I had him visited by Professor Albert Riva, of the University of Parma, and then Professor of the Principles and Practice of Medicine at the University of Perugia, who was so astonished at the result, that detecting no trace of the disease, he

doubted the correctness of the diagnosis. I insisted on the necessity of continuing the treatment, but the poor fellow being a waiter, was obliged to interrupt it to earn his bread. He had several relapses, but after each of them he resumed the treatment and at once improved. At the beginning of the current year he had another relapse; in May he entered this hospital, where he died the following August of pulmonary emphysema with phthisis, fourteen years and a half after the first treatment.

CASE I.—N.N. from Ispra, on Lago Maggiore, was 32 in 1885. She had suffered from cough for four years, and notwithstanding much treatment her disease persisted. I was there during the summer holidays and examined her in the middle of July; I found serious lesions at the apex of the right lung, with cavities; tubercular infiltration in the middle lobe, and in the left apex; daily fever, abundant perspiration. No examination of the sputum was made for bacilli. The woman knew she was consumptive and began at once uninterrupted inhalations, continuing them with perseverance. Seeing how much better she was for it, she never took off her mask, not even to go out; she was seen at church with the inhaler before her mouth. When I left Ispra (31st October) to return to Perugia, not only was there no fever, but the cough left her, and the local phenomena had subsided.

CASE II.—In the month of January, 1886, there came to me la Signora Rosa Scotti, aged 57, a native of Ponte Tresa. She had devotedly nursed a relative who had died of consumption at Bergamo. Three years after, when she was at Perugia, a tracheal tuberculosis developed, and in seven months went so far that when I examined her the malady had settled on the whole apex of the right lung extending to the middle lobe,

Case IV.—In August 1888, when I spent my summer holidays in the mountains of Lombardy, I was begged to visit il Signor C. C., aged 42, a railway clerk, who had gone to Trana, near Turin, because he was ill, and had become suddenly worse. I verified a very strong infiltration in the right lung with a hypophonesis so marked that it suggested a gathering of liquid in the pleural cavity, if the increase of the tactile vocal fremitus and some area of resonance at the base had not made the case clear. There were no cavities in any part of the diseased lung. Continued fever, 102° in the morning, 104° and even 105° in the evening, and perspiration more abundant than I had ever witnessed before. His father had died of consumption, and also a brother nine years previously whom he had nursed with assiduity. No examination was made for the bacilli in his abundant expectoration. He began immediately the uninterrupted inhalations. At the end of October, before returning to Perugia, I went to see him. The effect had really been startling. There was apparent on percussion a slight diminution of the resonance on the left, compared to that of the normal right lung. The fever and perspiration had ceased for a month and a half; almost no cough left, except some attacks in the morning. General healthy look, good appetite, easy digestion. The chief doctor of the railway department, the regretted Dr. Schina, was lost in wonder at such results. The patient continued the treatment, and the next year the recovery was perfect, so that Dr. Schina assured him he could then have insured his life, for no doctor could have perceived any sign of the illness he had had.

Case V.—N. N., veterinary surgeon, aged 28. He came to consult me in 1890. He had had several attacks of hæmoptysis, persistent cough, some fever, widespread infiltration at the right apex, with spots all over the lung on the same side, and also, but in less degree, on the left. He resolved to study as an apothecary, for he could not bear the fatigues of his calling. While studying pharmacy he adopted the inhalation treatment. He

recovered perfectly, bought a pharmacy, and enjoys perfect health as an apothecary at Assisi.

Case VI.—B. V., from Mantignana, near Perugia, was seen by me in April, 1893, with Dr. Busti, of Corciano, his own doctor, when he was eighteen years old. He had a large infiltration in the right lung, especially in the apex, and had been ill about a year. He followed the treatment for half a year, is now perfectly recovered, and is a student of pharmacy in this University.

Case VII.—R. A., aged 26, was born near Pinerolo. In 1895 he was transferred from the prisons of Turin to those of Perugia. He was a fine stalwart youth, and looked more than usually strong and hale. After some months the symptoms of consumption appeared, at first in the right lung, and were followed by a general deterioration of health. Presence of specific bacilli in the sputa. He began the treatment, and grew so rapidly better that in six months he was nearly recovered; but a tubercular arthritis manifested itself at the articulation of the right foot, and it was necessary to amputate the foot; Dr. De Paoli performed the amputation. He recovered perfectly, so that when he left this gaol in September, 1897, he looked as strong and hearty as before.

Case VIII.—A. X., aged 28, was treated by Dr. Bianconi, of Perugia, who informed her husband that the case was hopeless. A sister had died consumptive five years before. Her present malady had lasted two years. When I took her under my care in Nov., 1897, she was very thin, had no appetite, abundant perspiration, a cough with abundant purulent expectoration, had frequent and serious hæmoptysis, and tubercular infiltration at the apex of the left lung that spread as far as the fifth rib, with some excavation corresponding to the third space between the ribs, and some slight focus spreading to the middle lobe of the right lung. She was under treatment from Nov., 1897, to Nov., 1898. The change for the better was immediate; she had a miscarriage in June, 1898, then conceived again, and bore a

healthy child in March of this year. She had recovered so perfectly that it was impossible to detect any local symptoms. She spent some months at Gubbio, her native place, discontinuing the treatment, and returned to Perugia in an excellent condition; but last July she had a relapse. She resumed the inhalations at once, but though she took them intermittently, she actually feels very well now.

Case IX.—M. X., aged 15, was introduced to me by Dr. Virginio Blasi, in the beginning of 1898. She presented a tubercular infiltration at the apex of the right lung with cavities; daily fevers, sweatings, cough, extraordinary emaciation, etc. She adopted the treatment from January to September, when she seemed perfectly recovered. The local phenomena were quite gone, as Dr. Blasi repeatedly observed. She intended to spend the autumn with an aunt at Florence, but after a week returned because she was very ill. I was called in and found a serious pneumonia on the opposite side, a temperature of 106° and phenomena of acute endocarditis. She recovered from the pneumonia, and the apex of the right lung continued quite sound throughout the winter. In July I visited her again and found a relapse at the right apex. She resumed treatment and soon got better. Now she is well enough, though the endocarditis has left her with a very pronounced double aortic murmur.

Case X.—M. P., was imprisoned by mistake at the end of June, 1898, and while in prison I discovered in him a tubercular infiltration in the right apex with diffusion over the whole lung, and especially at the base. His wife had died of consumption the preceding year, and had previously been in the hospital owing to the breaking of a vomica into the pleura, which had produced pneumothorax with empyema. At the hospital his case was diagnosed as pulmonary tuberculosis. He was ghastly thin, with no appetite, bad digestion, and profuse perspiration. He began the treatment at once, and continues it to this day, but only during the night, though I have seen no trace of the disease for eight months.

Case XI.—C. A., entered the Infirmary of the Royal Female Refuge of Perugia, Aug. 11th, 1896, with widespread tubercular infiltration at the apex of the right lung. She used the inhaler till April in the following year, when she appeared perfectly recovered from her pulmonary consumption. But tubercular peritonitis set in, persisting until, in January of this year, an intestinal perforation was produced. She was three days in a critical condition, then began very slowly to improve. She lay in bed till May 21st, when she was compelled to leave the Refuge, having reached her twenty-first year. As she was unequal to the journey home I received her into the hospital, where I noticed clearly the remains of the disease at the apex of the right lung, soundly healed. After about two months she was better and went to her home in the neighbourhood of Naples, where she enjoys very good health, as I know from the nuns of the Refuge to whom she often writes.

Case XII.—U. G., a warder in the gaol at Perugia was my patient from June 28th last. He had a focus of disease at the apex of the right lung and another at the posterior portion of the base, he coughed much, expectorated abundantly, had sweats, a slight fever in the evening, and had become very thin, in short, the usual symptoms were present. I assured him that in three months he would be as good as cured. Indeed, on the 18th of September I presented him at the Umbria Medical Congress held at Perugia, with other patients, on the occasion of the discussion on tuberculosis, that he might be examined by the Congress, for the only symptom of pulmonary malady which he then showed was a jerky respiration at the seat of the lower focus. He is now fulfilling the duties of his calling at the gaol of Terracina, and enjoys very good health. I told him what he suffered from, that he might be induced to continue his treatment for a long time, though only during the night.

Cases XIII, XIV, XV, XVI.—In the Royal Refuge at Perugia are four girls under age, completely recovered

from pulmonary consumption by means of the uninterrupted inhalations. These are, A. P., who followed this treatment from May to July of last year, after which she went back to her companions without needing any further treatment; she enjoyed perfect health during the eighteen months; D. S., under treatment from May to December, 1898, has been for a year quite well, without need of further inhalations; D. S., took the inhalations from May 5th to the end of July in this year, and is also perfectly recovered; C. G., underwent treatment from September 7th, 1897 to April 25th, 1898. From April until now she has not presented the slightest symptom of pulmonary disease, though in August last she contracted typhoid which kept her in bed for forty-five days.

Cases XII.—XVI. have a special importance, for they show how easily the malady may be totally vanquished when treatment is begun at the very outset. These are some of the cases under my care during the past sixteen years, and I have quoted them because they may be easily verified, and because all present some interesting features.

In the gaols for men, where the inmates are compelled to remain twenty-three hours out of the twenty-four confined each in his own cell, and therefore under highly unfavourable conditions for treatment, the general results as shown by the registers of the infirmary have been as follows: Since 1889, 37 patients were treated with uninterrupted inhalations, *in different stages of the illness.* Of these 22 recovered, and 15 died.

The three following cases are the last, and though

they have but recently begun treatment, I consider that they may look forward to sure recovery.

Case XVII.—F. F., aged 24. No history of tuberculosis in the family. The present malady has lasted four years. Her physician is Dr. Vitaletti, of Colle del Cardinale (Perugia). In 1897 she was a fortnight in hospital at Perugia for the same disease. Some days after she left hospital she was visited by Professor Patella. She came to me on September 9th. She is a tall, pale girl. There was infiltration at the right apex with abundant râles, as well as at the base of the same lung, especially at the posterior portion. She began the inhalations on the same day. To-day, December 1st, her aspect is changed, pallor is gone, she has no cough, every trace of râles has disappeared, only the breathing is some what sharp.

Case XVIII.—R. F., aged 13. No history of tuberculosis in the family. The present illness dates four years back. Was a patient of Dr. Vitaletti above mentioned, and was also treated by Dr. Vincenzo Teyxeira. She is pale and thin. Abundant râles at the apex and at the base of the left lung. Frequent cough with abundant expectoration. Inhalations began on the same day that I examined her, August 15th (last). When I re-examined her, November 5th, I made on my register the following entry, "Impossible to see where she has been ill, a little coughing in the morning with slight expectoration."

Case XIX.—R.O., aged 23, from Perugia. No history of tuberculosis in the family. A year ago he had a fistula *in ano* while serving as a soldier, for which he was thrice operated on at the military hospital to no purpose. In March, the same year, he felt a pain in his left shoulder, with a cough. In April he entered the military hospital at Perugia, having a violent cough and abundant expectoration in which was found the specific bacilli. He was dismissed from military service in June, as disqualified by pulmonary consumption.

When I visited him at the beginning of July I found him emaciated, worn to a skeleton; he had a marked infiltration at the left apex, reaching to the third rib, an obstinate cough, and abundant expectoration. I at once prescribed uninterrupted inhalations, and advised an operation for the fistula, burning the tissues deeply with the thermo-cautery. The fistula healed in September. On seeing him again to-day, November 25th, I have written down, "No cough left, a healthy, sound appearance, grown much stouter; all signs of illness gone, except some occasional dry râles about the third rib on the left side."

Let us see now in detail how the treatment is to be conducted.

MODE OF TREATMENT.

We must never forget that the grand object is to combat the worst and only really dangerous enemy by rendering the tubercular lesions aseptic. Therefore our first care should be to arrange that our patients breathe, as far as possible, fresh air. And as in every case the air of inhabited rooms is always less pure by far than the outside atmosphere, we must insist on the necessity of having the patient out of doors as much as possible, notwithstanding that he has the mask on his face. The patient should live, when possible, in a country house, where he can spend the greater part of the day in the open air. If the season is cold and the patient feverish, it will be very beneficial to keep him the whole day in the open air on an invalid chair, well wrapped up, that he may not feel cold. He must be guarded against air rendered impure

by putrefactive organic substances. The exhalations arising from privies, dunghills, and from any impurities that may exist in the outbuildings, courtyards, etc., are highly pernicious. From the sinks in kitchens there often comes a nauseous smell which is always evidence of foul air.

For these reasons the greatest care is needed to ensure that the house is kept well aired, day and night, but especially at night. During the night the windows of the room should always remain open (open panes and closed blinds; and if the latter are absent, still open panes). This is of the greatest importance, even more than keeping windows open by day, for during the day the patient goes out of doors, or from room to room, but during the night he remains for eight or ten hours in the same room, which, however large, is vitiated by organic exhalations after two or three hours. Both doctor and patient should bear in mind that there exist in the lungs small lesions always ready to become septic by the introduction of impurities carried to them by the air, and though they are harmless as long as they are simply bacillary they become highly dangerous and deadly when thus infected. Bearing all this in mind, it is needless to dwell on details which each can fill in for himself, according to the cases that present themselves. I will however, make one comparison. In child-birth the placenta detaches itself from the

fundus of the uterus, leaving a physiological wound which heals of itself, and presents no peril if it does not become septic. The infection of this wound (as large as the palm of the hand) is not frequent by reason of the singular position it occupies. But when for any reason it does become septic a puerperal fever, and frequently death, follows in consequence. The 1,200 deaths which every year occur in Italy from puerperal fever are due solely to this cause. Now if we compare with this the facility with which the small lesions of a tuberculous lung may receive infection we shall realize the extreme necessity of air free from micro-organisms. It is exactly the same principle which, applied by Lister to surgery, caused the disappearance of the awful death rate formerly observed after surgical operations, and marked the commencement of modern scientific surgery; one might well say that real surgery did not exist previously. Applied to modern obstetrical teaching it immediately brought about so great a reduction in deaths from puerperal fever that whereas twelve years ago there were in Italy 3,000 a year, there are now only 1,200; and when the same principles are generally employed in the cure of pulmonary consumption, not only will each patient be placed in the most favourable condition for recovery, but the present frightful mortality from this terrible scourge will be similarly diminished.

It must not be imagined that because the mask is worn pure air may be disregarded. By its use there is, no doubt, a strong disinfection of the respiratory passages; but our first care should be to remove every means of infection—to be sure that new irritating agents do not reach the injured parts, although they might be destroyed later by the action of the remedy.

The resistance of micro-organisms to disinfecting substances is sometimes very strong, and their destruction often requires prolonged action. When a lung is much injured and contains numerous cavities, these contain a quantity of thick, purulent liquid which serves as a medium for the proliferation of septic germs, and is disinfected with more difficulty, for in it the disinfectant penetrates proportionately to its degree of solubility, and it is only accomplished by very prolonged action.

Let us remember that by wearing the mask we do not entirely avoid the bad consequences of breathing infected air.

DIET.

The three principal articles of food should be meat, eggs and milk, but beyond these anything the patient may fancy should be allowed him without restriction. The food, whatever it be, cannot harm provided it is rich in nutritive principles and is employed in such moderate quantities

as not to interfere with the appetite for more substantial food. The diet, therefore, should be very varied and abundant, and the meals should be rather frequent, eating little each time in order to make digestion easy. The desire for a particular food is sometimes produced by a real want of nature, and generally the patient digests well the food he relishes. Wine should be used in very moderate quantity, spirits forbidden, and smoking should also be abandoned.

EFFECTS AND DURATION OF THE TREATMENT.

The effects of the treatment vary according to the cases. Even with considerable lesions, while they are isolated from each other so that the inhalations may penetrate all the pulmonary tissue, considerable improvement may be noticed even within a month. I quote the following example as an illustration: –

During the recent medical meeting at Como my friend Dr. Beltramini begged me to visit Mrs. M. G., aged 29. My notes after an examination on October 6th are the following: Pleurisy at the age of 14. The first sign of tuberculosis two years ago after child-birth. She had then hæmoptysis and fever with cough. The cough has continued at intervals ever since, though it is of but little consequence. Blood-stained

expectorations have been repeated occasionally, especially five months ago. At present the general nutrition is fairly good, though the patient has lost flesh; abundant night-sweats; obstinate cough, especially in the morning, sometimes very bad with thick sputum; regular pulse. Considerable infiltration at the apex of the left lung; no cavities were detected; slightly sharp breathing, and hypophonesis at the base of the right lung outside the parasternal line.

On November 14 her husband wrote: "The night-sweats have disappeared, no cough, expectoration very slight, appetite good, increase of 3 kilograms in weight, consequent feeling of ease; these are the effects resulting after my wife has followed the treatment you prescribed, to which she has scrupulously adhered."

This is the general course of events. The fever subsides, even when it arises from large lesions, so long as the inhaled substances are able to reach and thus disinfect them, but if the lesions are so numerous and broken down that the pulmonary tissue is in places cut off from communication with the outside air, the improvement is much less marked. While the fever generally disappears in less than a month, under these conditions, in which local disinfection is difficult, it may require a considerably longer time.

The bacilli of tuberculosis frequently disappear

from the sputum in less than a month, but it is impossible to speak positively as to time. We should always remember that the character of pulmonary lesions is very varied. The bacilli which are commonly found in the respiratory organs, and those of the smaller lesions, which are easily reached by the remedy, certainly disappear rapidly. But when there are numerous and wide-spread cavities, very dense and difficult to disinfect, the bacilli may be found even after several months' treatment.

The rate of improvement observed in the first month is generally continued, until after some time the patients assume the normal appearance of health with good appetite and digestion, without cough, sweat, etc., so that they consider themselves recovered. *This is certainly the most dangerous period, for recovery is still far from complete, and if, as frequently happens at such a period, they give up the treatment, the consequence is a relapse.*

We must never forget the insidious character of this disease and the pathological conditions of the lesions. We know that it is produced by a bacillus of great resisting power whose spores possess even greater. By the action of the antiseptics they are prevented from multiplying, and are no doubt destroyed in great quantities; but some persist, for they are surrounded and enclosed in caseous material as in a capsule, and their spores very

probably do not die. Remembering the infinite variations in the lesions, this is easily understood. *Treatment must therefore be persisted in, even after the patients feel completely healed.* Even in slight cases treatment must never last less than a year.

When the second or third stage has been reached the cure will require much more time. In the great proportion of my cases it lasted about two, sometimes three, and even as long as four years.

One case required seven years. It was that of a woman in the gaol at Perugia, M. M., aged 40. I took her under my care when she was very ill, with pulmonary lesions on both sides; the lungs were infiltrated from top to bottom. She began the treatment in 1890 and continued it till 1897, when she was transferred to the prisons of Florence. From the latter she was sent to the gaol of the Giudecca at Venice. When she left Perugia the lungs were in better condition, but not wholly recovered. Lately, when at Venice, I went to see her and found her in the infirmary of the establishment. She had no fever, was not emaciated, had a good appetite and digestion; her aspect was good, but she had a cough and râles in various parts of the lungs.

We must, however, remember that this woman is a prisoner, and, except for a few hours a day, is forced to live in a confined space with other sick people during the whole time of her treatment

in the infirmary; and although the treatment was continued, it was easy for the lungs to be re-infected. I believe that patients who live in the open air need never continue the treatment for so long a period.

As to the results, I have the deepest conviction, founded on observation, that when treatment is scrupulously carried out in the manner indicated and continued several months after all local phenomena have disappeared, it leads certainly to the recovery of the patients in the first stage, and even in the second if the nature of the lesions is not such as to entirely cut off some part of the lung from every external communication as before described. A glance at the description of the preceding cases should be convincing on this point. Regarding the third stage, it is wise to take into consideration how far the lungs may have been destroyed; but even here, provided the lesions are such that they may be disinfected, all hope need not be relinquished. Last year I was called at Alassio to a lad of 19 in a desperate condition. I expressed to his father my opinion that the case was hopeless. However, the treatment was begun, and the effect for the better was so considerable that the father hoped for a while I had been mistaken. Unfortunately it did not prove so.

Seeing such results, I advise treatment even in desperate conditions. There is one great drawback

in the remedy being visible to the eye, for when there is a fatal termination the uninitiated may say the treatment is useless, and other patients may become distrustful. That patients, however, should not be forsaken even in the third stage is illustrated by this case: Two years ago an English lady living in Rome, who had been several years my patient, bade me examine her servant, a young man of 21. The latter had fever 102° in the morning and 104° in the evening, and such lesions in the whole of the right lung and at the apex of the left that I judged the case desperate, and at first declined ordering the inhalations, deeming these quite useless on account of the ravages of the disease. The lady would, however, have him adopt the treatment. This servant is a native of Castel del Piano in the province of Grosseto, and I sent him to his parents there. He followed my prescriptions faithfully, continued to improve, and now may be considered as good as recovered.* Dr. Benedetti wrote from Castel

* The following is the letter he wrote to me in all its simplicity:—

ROME, *October 5th*, 1899.

"MY GOOD DOCTOR,—I come to thank you for all the care you had of me, and I let you know that now I am come to Rome, as I hope the doctor of Castel del Piano has written to you. I have been six months without coughing, I feel strong, I have a good appetite, and I relish all I eat. I tell you I never felt so well as I do now. My family and I will always think of the miraculous mask and of the good care you had of me, and we will pray to God He grants you

CHAPTER V.

PREVENTIVE TREATMENT.

WHEREVER there is a consumptive patient there is peril of infection for all with whom he lives. The patient distributes bacilli in two ways—by expectoration and by the tiny drops of liquid he emits by coughing, sneezing, laughing, or speaking aloud. The bacilli thus emitted are a continual source of infection for the dwelling, therefore a peril to all in the house. Against expectoration one can guard by simple measures; the sick person must never expectorate except into his own spittoon, which is disinfected by boiling. So much has been said and written on this point that I have nothing to add.

Regarding the numerous bacilli sent forth into the air by the other acts mentioned, till now nothing has been suggested. By wearing the mask this danger is lessened in two ways: (1,) The respiratory channels are at once disinfected and the bacilli cease being emitted during these acts; (2,) Even should some bacilli occasionally escape from the mouth they are at once arrested by the sponge and immediately destroyed. Therefore the use of the mask is the best guarantee against danger for the

household. Lastly, I must call attention to a highly important fact. We have seen at the outset that when a second member of the family contracts tuberculosis it may be several years before the mischief it produces has gone far enough to trouble the patient and induce him to consult a doctor. Thus an individual may have been consumptive for three or four years, and even more, without being aware of it. In view of this I would advise the members of a family, where there is a consumptive patient, to wear the mask now and then as a precaution. Wear it, for instance, at night for a fortnight, then leave it off for a fortnight, then begin again, and so on. It is certain that the peril of infection in a family where there is a consumptive person is always considerable. The use of the mask may be considered superfluous when one is perfectly well ; but, remembering that consumption gives in the beginning no trouble at all, everyone will admit the importance of this preventive measure.

The use of the mask causes no disagreeable consequences, therefore I hope my earnest advice will be acted upon. Its immediate effects may not be easily visible, but the benefits that society might derive from its use as a preventive will be greater than those produced by uninterrupted inhalations as a remedy, by bringing about an extraordinary diminution in the number of cases of consumption.

CHAPTER XI.

CONCLUSION.

In bringing this small book to a conclusion, let me say that from the analysis of the facts given we may look forward to a day, perhaps not remote, in which tuberculosis of the lungs will, if it does not totally disappear, at least be very greatly reduced amongst us.

If we can induce the whole people to believe that by breathing pure air we maintain in normal conditions the great and powerful line of defence which we all possess in the epithelium that covers our respiratory organs; if they understand therefore how great the danger is for a whole family to dwell in confined rooms without air, light or sun, in which, moreover, there may be sources of continual poisoning through the foul exhalation from inside water-closets, kitchen sinks, etc., as well as from the corruption of filth outside, a great point will be gained. They will then seek for better ventilation by day and night, and will carefully remove all those causes that in the past have been, and even now are, the most fertile sources of tuberculosis. I have never met a person who,

though he at first submitted reluctantly to sleeping with open windows, ever afterwards repented of having done so; nay, I must add that all without distinction grew enthusiastic about it, seeing the great advantages derived from it. We never should endeavour to screen ourselves from cold by excluding pure air, but rather by adding clothing. In malarial districts, as it is now well ascertained that the mischief does not reside in the air but in the mosquitoes that inoculate with their sting, a mosquito-net should be nailed to the window frame, through which the gnats could not pass, and windows could be kept open.

Remembering that the epithelium keeps sound and performs its function if we breathe freely, not leaving any part of the lungs inactive, and that the apex is a very vulnerable point if the respiration is tardy or in any way impeded, we see how we may prevent the bacillus of tuberculosis from settling on the bronchial mucosa, and we have here a guide showing us how to avoid the malady when there is a consumptive member in the household. In such cases the disinfection of the sputum, ample free ventilation and abundance of light are not only the best precautions against infection, but themselves constitute the most efficient aid towards treatment for the patient himself. If, further, the members of the family use the mask occasionally, say during the night only, they

may defy infection. The most important point is that now we must act on principles exactly contrary to those which have prevailed in the past, and remember that the precautions formerly taken to exclude air lest we should catch a cold, are precisely the means of putting ourselves in the most favourable conditions for contracting tuberculosis.

In the consumptive himself I would inculcate this great fact, which I have tried to emphasize throughout this little book: Let him remember that it is not the tuberculosis that kills, but the infection of the tubercular tissues by inspiration of foul air.

From this statement of facts I think that the present generation may look forward to an epoch not very remote in which, if the people become more enlightened, pulmonary tuberculosis will be as uncommon as through antiseptic surgery the septicæmias, the pyæmias, the nosocomial gangrenes now are in our hospitals; and that this may come as soon as possible is the most earnest wish of my heart.

APPENDIX.

MEAT AND MILK OF TUBERCULOUS COWS AS SOURCES OF TUBERCULOSIS IN MAN.

THE last sheets of this book were passing through the press when Prof. Koch read his paper at the "British Congress on Tuberculosis" (July 23rd, 1901), in which he contended that meat and milk from tuberculous cattle cannot produce tuberculosis in man.

This assertion was not favourably received by the majority of the Congress; in fact many thought that it was the result of imperfect experiment and observation. Two days afterwards Prof. John Macfadyean read at the same Congress a very able paper which sought to controvert Dr. Koch's claim, and his assertions were accepted with general approval by the numerous assembly.

The author of this book held the same opinion as that expressed by Prof. Macfadyean until about two years ago. But the observation of some striking facts then convinced him that bovine tuberculosis could never be communicated to man through tuberculous meat or milk, so much so that in a paper published by him in the *Salute Pubblica* (April 15th, 1901, p. 102), he arrived at the conclusion that milk from tuberculous cows is not able to produce intestinal tuberculosis in human beings.

Prof. Koch said that, as the result of his experiments, human is quite different from bovine

tuberculosis. "A number of young cattle which had stood the tuberculine test, and might therefore be regarded as free from tuberculosis, were subjected to infection in various ways with pure cultures of tubercle-bacilli taken from cases of human tuberculosis; some of them got the tubercular sputum of consumptive patients direct. In some cases the tubercle-bacilli or the sputum were injected under the skin, in others into the peritoneal cavity, in others into the jugular vein. Six animals were fed with tubercular sputum almost daily for seven or eight months; four repeatedly inhaled great quantities of bacilli, which were distributed in water, and scattered with it in the form of spray. None of these cattle (there were nineteen of them) showed any symptoms of disease, and they gained considerably in weight. From six to eight months after the beginning of the experiments they were killed. In their internal organs not a trace of tuberculosis was found. . . ."

"The result was utterly different, however, when the same experiment was made on cattle free from tuberculosis, with tubercle-bacilli that came from the lungs of an animal suffering from bovine tuberculosis. After an incubation period of about a week the severest tubercular disorders of the internal organs broke out in all the infected animals. It was all one, whether the infecting matter had been injected only under the skin or into the peritoneal cavity or into the vascular system. High fever set in, and the animals became weak and lean; some of them died after a month and a half to two months, others were killed in a miserably sick condition after three months. After death extensive tubercular infiltrations were found at the place where the injections had been made, and in the neighbouring lymphatic glands, and also far ad-

vanced alterations of the internal organs, especially the lungs and the spleen. In the cases in which the injection had been made into the peritoneal cavity the tubercular growths, which are so characteristic of bovine tuberculosis, were found in the omentum and peritoneum. In short, the cattle proved just as susceptible to infection by the bacilli of bovine tuberculosis as they had proved insusceptible to infection by the bacilli of human tuberculosis. . . ."

The difference between human and bovine tuberculosis appeared not less strikingly in similar experiments made with pigs, apes, sheep and goats.

"Considering all these facts I feel justified in maintaining that human tuberculosis differs from bovine, and cannot be transmitted to cattle. . . . But, now, how is it with the susceptibility of man to bovine tuberculosis? This question is far more important to us than the susceptibility of cattle to human tuberculosis, highly important as that is too. It is impossible to give this question a direct answer, because, of course, the experimental investigation of it with human beings is out of the question. Indirectly, however, we can try to approach it. It is well known that the milk and butter consumed in great cities very often contains large quantities of bacilli of bovine tuberculosis in a living condition, as the numerous infection-experiments with such dairy products have proved. Most of the inhabitants of such cities daily consume such living and perfectly virulent bacilli of bovine tuberculosis, and unintentionally carry out the experiment which we are not at liberty to make. If the bacilli of bovine tuberculosis were able to infect human beings, many cases of tuberculosis could not but occur amongst the inhabitants of great cities."

These are the expressions of Koch, which, as he says, have one weak point: The wanting of the proof by direct experiment that bovine tuberculosis is not communicable to man.

Now I think, as I mentioned before, that, by a quite different way and by the observation of other facts, I could prove that neither meat nor milk of tuberculous cattle can give tuberculosis to man. The fact that Prof. Koch arrived at the same conclusion by laboratory experiments gives to the solution of the problem all the evidence of truth and certainty.

Let us consider the following statistical data given by the last "Annual Report of the Registrar-General" of England, regarding the three principal forms of tuberculosis, Intestinal tuberculosis or Tabes mesenterica, Tubercular meningitis, and Lung tuberculosis or phthisis, as they occurred in 1899 at different periods of life.

MORTALITY IN ENGLAND AND WALES IN 1899, AT DIFFERENT PERIODS OF LIFE, FROM—

AGE	TABES MESENTERICA	TUBERCULAR MENINGITIS	PHTHISIS
0 to 1 year ...	3,208	1,820	431
1 to 2 years...	1,100	1,228	394
2 to 3 " ...	366	712	221
3 to 4 " ...	164	478	134
4 to 5 " ...	106	372	131
5 to 10 " ...	325	917	576
10 to 15 " ...	186	353	1,011
15 to 20 " ...	166	176	3,185
20 to 25 " ...	133	102	4,784
25 to 35 " ...	190	140	4,955
35 to 45 " ...	98	44	6,718
45 to 55 " ...	41	15	3,468
55 to 65 " ...	19	4	1,357
65 to 75 " ...	4	1	187

These figures differ but very little from those of preceding years, in which we always find the following characteristics: (1,) The largest number of deaths during the first years of life is given by tabes mesenterica, then by tubercular meningitis, and lastly by phthisis. (2,) In all the three forms of tuberculosis the largest number of deaths is observed during the first year of life; we have a diminution during the second year, and this diminution rapidly increases during the following years, so much so that during the fifth year of life all the three forms of tuberculosis have a very low death-rate. (3,) From this period of life, while the first two forms continue to decrease for all the succeeding years of life, the third form (phthisis) begins to increase until it reaches its maximum in from 25 to 35 years, diminishing slowly after that time.

These facts can be better observed in the following diagram (Fig. 12), where the three forms of tuberculosis are represented each by a different line, ——— tabes mesenterica; – – – – – tubercular meningitis; and ..... phthisis. The height of each line at different periods of life represents the monthly death-rate of each period.

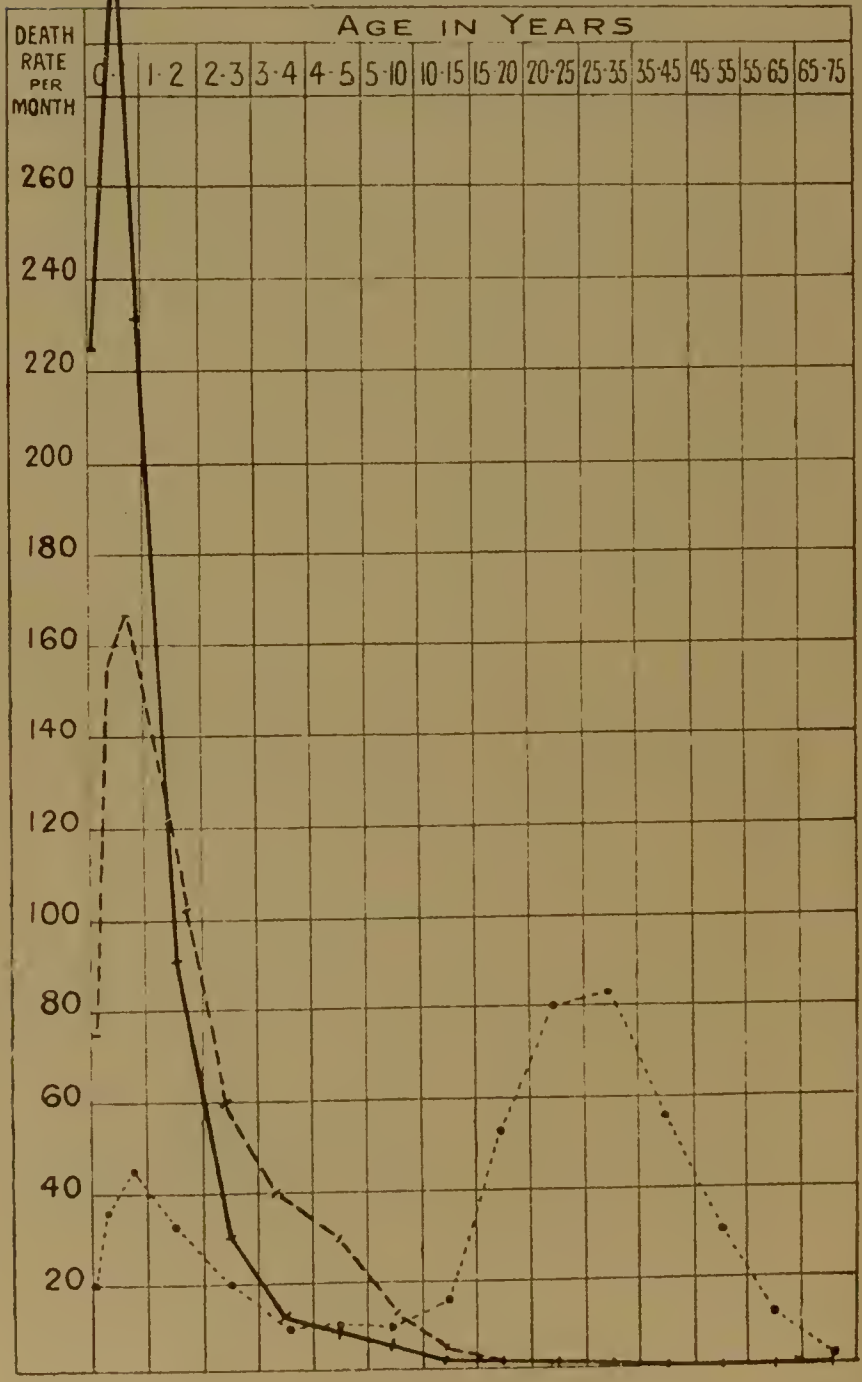
By these data we discover that *tuberculous meat* cannot be a source of tuberculosis in man, as practically we have no tabes mesenterica during the periods of life when we eat the largest amount of meat. Indeed, we see that tabes mesenterica is by far the highest during the first year of life, when no meat at all is used as a food.

We can see, too, that *tuberculous milk* cannot be a source of tuberculosis in man. My much regretted friend, Sir Richard Thorne Thorne, medical officer to the Local Government Board, in his admirable Harben Lectures (1898), attributed the high death-

FIG. 12.

CHART SHOWING THE RELATIVE MORTALITY AT VARIOUS PERIODS OF LIFE, FROM —

——— *Tabes mesenterica* : – – – *Tubercular meningitis* ; . . . . *Phthisis*.



rate from tabes mesenterica during the first and second years of life to the influence of tuberculous milk. But we have numerous facts which prove that this cannot be. As we have seen, intestinal tuberculosis is practically confined to the first and second years of life. But we drink milk during all the other years of our life; why then must we have such a high mortality from tabes mesenterica during the first years of life, and not afterwards?

Besides that, we know, and we all agree with Prof. Macfadyean, that "tuberculosis is a disease that develops slowly." This slowness of development is quite in opposition to the facts under our consideration. If milk was the cause of the high death-rate in infancy for tabes mesenterica, this slowness of development should produce the highest mortality during the second and the third year, and not during the first year. Not only so, but we see that during the first months of life the death-rate is as high as the death-rate of the eleventh or twelfth month. From 0 to 3 months in 1899 the number of deaths from tabes mesenterica in England and Wales has been 681 (227 per month); 1,142 from 3 to 6 months (380 per month); and 1,385 from 6 to 12 months (230 per month). It is impossible to admit that during the first month of life, when cow's milk is by far less used than during the tenth, eleventh, or twelfth month, and when the milk had not time to produce mortal intestinal changes, the deaths should be as numerous as in the tenth, eleventh, and twelfth month.

Again, how can we explain the high number of deaths from tubercular meningitis, which cannot certainly be produced by the ingestion of tuberculous milk? Perhaps one might say that the large number of deaths from tubercular meningitis

are not primary cases, but secondary to intestinal tuberculosis. This cannot be, because from the slowness of development of the disease we cannot have so much tubercular meningitis during the first months of life, *secondary* to intestinal tuberculosis. We must observe, too, that during the second year of life the number of deaths from meningitis is larger than that from tabes mesenterica, so that the secondary cases would be more numerous than the primary ones.

All these facts provide the strongest evidence that milk has not the least influence on the high mortality from tabes mesenterica during the first years of life

After giving expression to some of these views at the London Congress, Prof. Macfadyean in his paper, wherein he strove to refute the assertions of Prof. Koch, made an allusion to these statistical data. He at first referred to what Sir Richard Thorne had said in his Harben Lectures, and afterwards stated: "There are several weak points in this argument (the argument of Sir Richard). Perhaps the weakest of all is the assumption that the deaths certified under the head of *tabes mesenterica* correspond closely with those which the pathologist would classify as cases of primary alimentary infection. It is scarcely possible to doubt that the term *tabes mesenterica* in the Registrar-General's returns covers a heterogeneous collection of cases, of which the majority may not be cases of tuberculosis at all. But if even it is agreed to accept all the cases registered under the head of tabes mesenterica as instances of primary alimentary infection, the figures found do not support the contention that milk is responsible for all the cases of *tabes*. Now, if tuberculous milk were a frequent cause of tuberculosis, one would

not have expected the death-rate from that cause to decline among children between one and four years of age, for there is no reason to suppose that there has been any decline in the use of cow's milk in the feeding of children at that age during the last fifty years. The fact appears to be that the Registrar-General's returns do not afford much trustworthy information with regard to the number of cases of primary alimentary tuberculosis, and are absolutely worthless as an indication of the extent to which human beings are infected by means of milk."

As we have already seen, the only period during which primary tuberculosis is found is in the early years of life, and particularly during the first, second, and third years. During all my long experience I have never found in adults a case of primary intestinal tuberculosis. Prof. Osler says that it is very rare, "occurring in but one instance in 1,000 autopsies upon tuberculous adults in the Munich Pathological Institute." This is quite in accordance with the quoted returns of the Registrar-General, which show that after the fifth year of life intestinal tuberculosis is practically *nil*. Not only so, but it is most probable that the few cases of deaths in the adults reported as tabes mesenterica are in fact cases of primary pulmonary tuberculosis, certified after death as tabes, because they died with the prevailing symptoms of tabes.

Now if we accept the assertions of Prof. Macfadyean that "the figures found in the Registrar-General's returns do not support the contention that milk is responsible for all the cases of tabes," and that the "Registrar-General's returns do not afford much trustworthy information as an indication of the extent to which human beings are infected by means of milk," we must necessarily

ask where shall we find the trustworthy indications for the alleged harm produced by tuberculous milk in human beings?

Of course we cannot say that the Registrar-General's returns about tabes mesenterica must all be accepted as cases of primary alimentary tuberculosis, but it is certain that they are quite near enough to the truth. This is proved by the fact that the returns of all the causes of death in different countries agree with the English. In Italy in 1890 we had the following returns* :—

Age	Tabes Mesenterica	Tubercular Meningitis	Phthisis
0 to 5 years...	7,784	3,731	1,463
5 to 10 ,, ...	711	738	707
10 to 20 ,, ...	600	456	4,745
20 to 40 ,, ...	712	257	10,438
40 to 60 ,, ...	425	91	6,716
60 to 80 ,, ...	387	41	1,967

The Austrian and Prussian returns unite all the forms of the disease, but they are equally expressive, as they have the same characteristics that we noticed in the English. The Austrian returns are particularly interesting, as they give the separate deaths for every month during the first six months of life.

Deaths from Tuberculosis in Austria in 1896.

1st month ...	724	2nd year,	4,767	18 to 20 years,	6,296
2nd ,, ...	687	3rd ,,	2,694	20 to 30 ,,	16,112
3rd ,, ..	737	4th ,,	1,515	30 to 40 ,,	12,872
3 to 6 months	1,985	5th ,,	1,117	40 to 50 ,,	10,085
6 to 9 ,,	1,696	6th ,,	878	50 to 60 ,,	9,512
9 to 12 ,,	1,653	6 to 10 yrs.	2,865	60 to 70 ,,	6,543
		10 to 15 ,,	3,227	After 70 ,,	2,267
Total 1st year	7,482				

* I quote the returns of 1890 because in the subsequent years all the forms of tuberculosis are grouped together.

Remembering that the high death-rate during the first years of life is principally produced by tabes mesenterica and tubercular meningitis, we see here how high it is even during the first, second and third months; we see too a diminution during the second year, as in the English returns, a diminution which is by far greater during the following years until the tenth, when the increase begins to be produced by pulmonary tuberculosis, exactly as we noticed in the English returns.

As all the statistics I have consulted present the same characteristics as those now described, we must accept as facts that during the first year of life we have a very high mortality produced by intestinal tuberculosis, and that tuberculous milk has not the least influence in such a high mortality, as it has none on tubercular meningitis, which comes next to tabes mesenterica.

Prof. Koch in his paper said that cases of "so-called" primary tuberculosis of the intestine are extremely rare. "Among the great *post mortem* material of the Charité Hospital in Berlin, ten cases of primary tuberculosis of the intestine occurred in five years. Among 933 cases of tuberculosis in children at the Emperor and Empress Frederick's Hospital for Children, Baginsky never found tuberculosis of the intestine *without simultaneous disease of the lungs* [italics are mine] and the bronchial glands. Among 3,104 *post mortems* of tubercular children Bisdert observed only sixteen cases of primary tuberculosis of the intestine."

Contrary assertions cannot disprove such a large amount of facts as are observed in all the different countries of Europe, where the statistical returns agree so strikingly! I think that in these *post mortems* the tendency has been to consider as secondary cases of intestinal tuberculosis, all the

cases where a tubercular infiltration has been found in the lungs. But the general practitioner, who has observed the beginning of the disease in the intestine, and the prevailing intestinal symptoms as the cause of death, cannot say the same thing. Anatomically we know how easily the tubercular bacilli can be transported to the lungs; and it is quite natural that the greatest number of cases of primary intestinal tuberculosis in the latest stage of the disease present a tubercular infiltration in the lungs, which induced the pathologist to consider them as secondary cases, while the general practitioner certified them as tabes mesenterica.

That this is the correct explanation is shown from the fact that during the last few years the evidence from the *post mortem* records of two of the largest hospitals for children in Great Britain has been analysed with great care, in order to see what evidence they afforded as to the relative frequency of the different methods of infection in tuberculosis. In the case of the Hospital for Sick Children in Great Ormond Street, this has been done by Dr. G. Still, and in the case of the Royal Hospital for Sick Children in Edinburgh by Dr. Shennan. The conclusion at which Dr. Still arrived was that 29·1 per cent. of the cases of tuberculosis in children had primary infection of the intestine; and Dr. Shennan arrived at an identical conclusion, estimating at 28·1 per cent. the cases of primary intestinal tuberculosis among children in Edinburgh. "There does not appear to be any ground for supposing that there is a large margin of error in these statistics, as the number of cases dealt with was considerable (547 in two series), and in both the *post mortem* appearances were interpreted in a way to which no exception can be taken. In face of these statistics it is not

possible to assent to the statement that cases of primary tuberculosis of the alimentary canal are extremely rare. Precisely the contrary conclusion is the one that must in the meanwhile be drawn with regard to the state of affairs in this country." (Macfadyean.)

The only possible explanation of these figures seems to me to be the following: Their almost absolute uniformity justifies the assumption that they represent as many deaths of true tuberculosis. And then we must ask, How is it that during the first year of life the death-rate is by far higher than at any other period? How is it that although tuberculosis is so slow in its development it kills more persons during the first year, nay, even during the first month, than at any other period? Is it possible that a new-born babe in a month has sufficient time to be attacked by the external bacillus, and to be killed by this slow-working micro-organism, in such a manner, although at no other period of life (during which there is a much longer time for the external bacillus to act) is the mortality so high as during the first month? No one could admit this, whatever may be the mode of entry of the bacillus.

These numerous deaths are due to the fact that infants contract tuberculosis in the maternal uterus from tuberculous mothers. If they are deeply and extensively infected they die before being born; if less they die during the first month, or the second, or the third, and so on. As we proceed in life the influence of heredity rapidly diminishes, as is shown by the rapidly descending lines in the diagram (p. 134), so much so that at the third year of life it is already very low. The two essentially hereditary forms (tabes mesenterica and tubercular

meningitis) continue to be extremely low during the whole life, while after the fifth year of age the pulmonary form, the only one that man can acquire externally by inhalation of the bacillus, begins to increase in the manner indicated.

We have another disease which behaves in the same manner as tuberculosis, that of syphilis.

That syphilis is hereditary has been known to medical science for several centuries. If we examine it to see how it behaves in the different periods of life, we are struck by its resemblance to tuberculosis.

DEATHS IN ENGLAND AND WALES FROM SYPHILIS (1899).

0 to 3 months,	712	4 to 8 years,	2	35 to 45 years,	134
3 to 6 „	311	5 to 10 „	6	45 to 55 „	108
6 to 12 „	176	10 to 15 „	7	55 to 65 „	68
1 to 2 years,	57	15 to 20 „	11	65 to 75 „	25
2 to 3 „	13	20 to 25 „	30	75 to 85 „	6
3 to 4 „	6	25 to 35 „	21		

We see here the *period of inheritance* well marked, particularly during the first year of life, as in tuberculosis, and then a rapid diminution. We have then a *period of quiescence*, like that of tuberculosis, when the hereditary influence has ceased and the influence of the external cause had not yet time to act; and at last we have the *period of acquired syphilis*, which, like the period of acquired tuberculosis, reaches its maximum at the age of about 35 years and then slowly decreases.

In conclusion I would say that during the first years of life all the forms of tuberculosis are inherited; tabes mesenterica gives the largest number of cases, because the intestine affords the largest surface to be attacked in the intra-uterine

life by the bacillus of a tuberculous mother ; next comes tubercular meningitis, because the surface presented by the meninges is not so large ; and afterwards the pulmonary form, as the lungs being unexpanded are very small in the fœtus.

In later years all the forms of tuberculosis are acquired, but only in one manner—by inhaling the bacillus of tuberculosis, producing pulmonary tuberculosis. All the other forms are then secondary to pulmonary tuberculosis, although it may happen that the primary form does not develop, while the secondary form develops to such a degree as to be the only cause of death.

These considerations make it clear that milk from tuberculous cows never produces tuberculosis in man, but that in the first years of life tabes mesenterica, as well as the other forms of tuberculosis, are always inherited ; and in adult life we never have primary intestinal tuberculosis.